PROGNOSTIC SIGNIFICANCE OF ARRHYTHMIAS IN ST ELEVATION MYOCARDIAL INFARCTION

Dissertation submitted in partial fulfillment of regulation for the award of M.D. Degree in General Medicine

(Branch I)

The Tamilnadu
Dr. M.G.R. Medical University
Chennai
March 2009

Coimbatore Medical College

Coimbatore - 641 014

PROGNOSTIC SIGNIFICANCE OF ARRHYTHMIAS IN ST ELEVATION MYOCARDIAL INFARCTION

Dissertation submitted in partial fulfillment of regulation for the award of M.D. Degree in General Medicine (Branch I)

The Tamilnadu Dr. M.G.R. Medical University

Chennai

March 2009

certificate

*This is to certify that the dissertation entitiled "***PROGNOSTIC SIGNIFICANCE OF ARRHYTHMIAS IN ST ELEVATION MYOCARDIAL INFARCTION** *" , herewith submitted by* **Dr.M.SELVAGANESH ,** *post graduate in General Medicine Coimbatore Medical College Hospital is the record of a bonafide research work carried out by him under our guidance and supervision from July 2006 to June 2008.*

Prof. Dr. JAMBULINGAM M.D

Unit Chief

Prof. Dr. K.UMAKANDHAN M.D

Professor & Head
Department of Medicine

Dean

Coimbatore Medical College

Coimbatore - 641 014

Date:

Coimbatore:

Coimbatore Medical College

COIMBATORE, TAMILNADU, INDIA - 641 014

(Affiliated to The T.N. Dr. MGR Medical University, Chennai)

ETHICS COMMITTEE

CERTIFICATE

Name of the Candidate	:	Dr. M. Selvaganesh
Course	:	M.D. (General Medicine)
Period of Study	:	2006 - 2009
College	:	Coimbatore Medical College
Dissertation Topic	:	Prognostic Significance Of Arrhythmias In ST Elevation Myocardial Infarction

The Ethics Committee, Coimbatore Medical College has decided to inform that your Dissertation is accepted / ~~Not accepted~~ and you are permitted / ~~Not Permitted~~ to proceed with the above Study.

Coimbatore - 14.

Date : 5.3.2008

Secretary
Ethics Committee

DECLARATION

I solemnly declare that the dissertation titled **"PROGNOSTIC SIGNIFICANCE OF ARRHYTHMIAS IN ST ELEVATION MYOCARDIAL INFARCTION"** was done by me from July 2006 to June 2008.under the guidance and supervision of **Professor Dr. P. JAMBULINGAM M.D.,**

This dissertation is submitted to the Tamilnadu Dr. MGR Medical University towards the partial fulfillment of the requirement for the award of MD Degree in General Medicine (Branch I).

Place : **Dr. M. SELVAGANESH**

Date :

ACKNOWLEDGEMENT

I express my gratitude to **Prof Dr. V. Kumaran,** the **Dean** Coimbatore Medical College Hospital for providing facilities to carry out this project work successfully.

I am extremely thankful to Prof. **Dr.UMAKANTHAN. K MD** Head of the Department of General Medicine and my unit Chief **Dr.P.JAMBULINGAM M.D** for their valuable guidance, support and advice rendered in completion of my project.

I express my sincere thanks to Prof. **Dr. D. Muthukumaran MD**, **DM**, HOD of Cardiology and. **Dr. D. Dharma Rajan MD. DM**., Asst. Prof of department of Cardiology for their valuable help throughout the course of my project.

I am extremely grateful to **Prof Dr.Yashodara** former Head of Department of medicine who helped and guided me in the study. I thank **Prof Neelambikai.MD**, Professor of Physiology and Head of Ethics Committee Coimbatore Medical College for the invaluable suggestions and corrections.

I extend my special thanks **Dr. Kumar Natarajan MD., Dr.P.Vishnuram MD., Dr.P.S.Rani MD., Dr. Arul MD**., Asst Professors of Medicine for their constant encouragement and support to carry out this study.

I whole heartedly thank the patients who took extra burden for the success of my study

INDEX

INTRODUCTION

Coronary artery disease is the leading cause of death globally. In 2001 coronary artery disease accounted for 7.1 million deaths world wide.[1]80% of which were in low income countries like India[2]. It has been estimated that by 2010, 60% of world's heart disease are expected to occur in India.[3] Indians are prone to get coronary artery disease at an earlier age than do people in developed countries because of the high prevalence risk factors like diabetes and hypertension[4,5]

Coronary artery disease is classified into stable angina and acute coronary syndrome. Acute coronary syndrome(ACS) includes ST segment Elevation Myocardial Infarction (STEMI), Non ST segment Elevation Myocardial Infarction (NSTEMI), and unstable angina.[6,7]

In Indian population ST segment elevation myocardial infarction is the most common type of acute coronary event and contributes to 60.6% of overall incidence of acute coronary syndrome.[8] The overall mortality in STEMI is approximately 4 to 7 % or even less in the published clinical trials[9,10]. However this is not the case in the real world situation.[11,12] This is because the patients enrolled in the randomized trials are selected ones and represented low-risk subgroup. Therefore the results of these trials are not applicable to 50% of patients in clinical practice[13]. A realistic view can be obtained from registry data. In India, CREATE registry data recorded in-hospital mortality rate of 7.9% and 30 day mortality rate of about 8.6%[8], which included both patients with unstable angina and AMI. V.Jacob Jose and Satya N. Gupta from Vellore, (Tamilnadu)

observed 16.9% in hospital mortality rate among South Indian population following STEMI.[14]

Almost 80 - 90 % of patients develop any one type of rhythm abnormality after acute myocardial infarction. The spectrum of arrhythmias following AMI, include ventricular arrhythmias (the major source of sudden death), conduction system disturbances, and atrial arrhythmias.[15] Arrhythmias are one of the most common cause of in-hospital, early (<30 day) and late mortality and morbidity following acute coronary syndrome. Arrhythmias influence the outcome directly or indirectly by their effect on the myocardium and hemodynamic status of the individual .Many studies have analyzed the influence of different types of arrhythmia individually, but data from India is scarce. In this study we have analyzed incidence and prognostic significance of entire spectrum of arrhythmia in a cohort of patients with acute ST segment elevation myocardial infarction.

REVIEW OF LITERATURE

CORONARY ARTERY ANATOMY :

The cardiac myocardium is supplied by the two coronary arteries arising from the aortic sinuses. 1. Left coronary artery 2. Right coronary artery. Knowledge about the areas supplied by the coronaries helps to correlate the occluded vessel with the region of the ventricular wall suffering from infarction

The left coronary artery originates from the left sinus of valsalva , and bifurcates into left anterior descending artery , left circumflex artery. Left anterior descending artery gives a branch to the interventricular septum, (septal perforating artery) and diagonal branches to supply anterolateral wall of the left ventricle. Left circumflex artery provides atrial branches to left atrium and marginal branches to supply lateral wall of left ventricle.

The right coronary artery originates from the right sinus of valsalva and supplies the right atrium, right ventricle and the inferior aspect of the left ventricle posterior aspect of interventricular septum. The endocardial layer of the heart receives supply directly from ventricular cavity. Subendocardial region is the least perfused zone of myocardium, therefore most susceptible to ischemia.

The dominant artery is generally defined as the one that provides the posterior descending artery to supply the posterior wall. In 85% of the population right coronary artery is the dominant. In the remaining, 10% of individuals will have left circumflex artery as dominant , the rest of the 5% will have codominance.

MYOCARDIAL INFARCTION :

Pathologically myocardial infarction is defined as ischemic necrosis of myocardium caused by the occlusion of the coronary arteries and its branches. It can be divided into transmural , subendocardial infarct according to the extent of myocardial involvement.When the necrosis involves full or nearly full thickness of the ventricular wall in the distribution of single vessel it is called as transmural infarct. In contrast subendocardial infarct constitutes area of necrosis limited to the inner one third or at the most half the thickness of ventricular wall.[16]

Coronary atherosclerotic disease is the underlying substrate in nearly 80% of the patients with myocardial infarction.[17].The initiating event is rupture of the atherosclerotic plaque which results in occlusive intracoronary thrombosis[18]. If an occlusive thrombus forms, the patient will develop transmural infarction with ST segment elevation in the ECG. On the other hand if the thrombus is non occlusive one, the patient will develop unstable angina or non ST segment elevation myocardial infarction because of less than transmural involvement.Ionic changes occurring in the dying myocardial cell result in electrical complication, contractile dysfunction (mechanical complication) loss of muscle mass, thinning of myocardium and increase the risk of complications.

ELECTROCARDIOGRAPHY:

Electrocardiography is one of the essential tools in the diagnosis of CAD. Not only for the diagnosis, it also helps to estimate the amount of myocardial damage and to

predict the prognosis following an infarction .Moreover ECG is the primary tool in arrhythmia analysis (other than this, ***Electro Physiological Study*** is the only modality, which can record electrical activity from several regions of the heart with its invasive intracardiac catheters and it is more definitive too). Initially, a 12-lead ECG is recorded. In addition, a long continuous recording using the lead that shows distinct P waves is often helpful for closer analysis; most commonly, this is one of the inferior leads (II, III, aVF) and occasionally V_1 or aVR. The ECG obtained during an episode of arrhythmia may be diagnostic by itself, obviating the need for further diagnostic testing.

ANATOMY OF THE CONDUCTION SYSTEM[19] :

This specialized system of heart constitutes sino atrial node, internodal (intra atrial) pathway, atrioventricular node (AV node) His bundle , right and left bundle branches, purkinje tissue.

Sino atrial node is located at the junction of superior vena cava with right atrium. Atrioventricular node is located in the right posterior portion of inter atrial septum. The internodal pathways include Bachman, Wenkebach, Thorel, which connect the sinus node and AV node.These are bundles of atrial fibres. Atrioventricular node is the only conducting pathway between atria and ventricle. It is continuous with bundle of His which gives off left bundle branch at the top of interventricular septum and continues as right bundle branch. The left bundle branch divides into anterior and posterior fascicle. These fascicle run subendocardially on either side of septum, come in contact with purkinje system, whose fibres spread over all parts of ventricular myocardium.

ELECTROPHYSIOLOGY OF CONDUCTING SYSTEM:

Myocardial cell in the resting state has membrane potential of approximately "-90 mv" called as polarized state. Influx and efflux of Na, K^+ , Ca^{++} ions in and out of the cells result in depolarization and repolarization thereby produce characteristic ***action potential curve*** , which has 5 phases.[20]

1- phase 0 – depolarization – rapid influx of sodium

2- phase 1 - early rapid repolarization – closure of sodium channels

3- phase 2 – plateau phase – Efflux of potassium. chloride ,influx of calcium ions

4- phase 3 - final rapid repolarization – efflux of potassium ions

5- phase 4 – resting membrane potential

Atrial and ventricular myocytes as well as purkinje fibres have the above said type of action potential called as fast channel action potential. Genesis of this type of action potential requires stimulation and can not occur spontaneously.

Automaticity :-

Sino atrial node (SA), atrioventricular node (AV), atrioventricular ring, coronary sinus opening area, and Purkinje fibers have special type of action potential called slow action potential. This action potential has shorter duration, characteristic feature in phase 4 that is spontaneous decay of resting membrane potential. This slow diastolic

depolarization (pacemaking potential) is resulting from T type calcium channel, when the resting membrane potential reaches the threshold value, the spontaneous depolarization occurs[21]. This electrophysiological property is responsible for the automaticity of these tissues. Eventhough a number of pacemaker cells are present, the sino atrial node dominates over others because of its high frequency discharge. Action potential in these automatic fibers is slow channel type. It has short duration, slow depolarization during phase 0, no plateau phase, but longer refractory period.

Conductivity:-

Depolarization generated in the sino atrial node spreads radially through the atrial myocytes as if they were syncitium, which is due to the presence of gap junctions between the myocytes. The rate of conduction through these fibers is a function of its membrane responsiveness defined by the rate of rise of action potential during phase 0. The fibers having past channel action potential conducts faster than fibres having slow channel action potential because rapid rate of rise of action potential during phase 0.[22]

Impulse generated in the SA node reaches the AV node after activating the atria. Before the excitation spreads to the ventricles a delay of about 0.1 sec occurs at the AV node called as AVnodal delay. From there impulse reaches the His bundle at the top of the septum, then it spreads to all parts of the ventricular myocardium through the rapidly conducting purkinje system fibers.

VASCULAR SUPPLY OF THE CONDUCTION SYSTEM:

Sino atrial node is supplied by the proximal branches of right coronary artery in 55% of individuals. In the remainders, it receives supply from the proximal branches of the left circumflex artery. Atrioventricular node receives its supply from the distal branches of the right coronary artery in the 90% of population. In the remaining 10%, it receives from the left circumflex vessel. The Right bundle branch is supplied by septal perforators. The anterior division of the left bundle branch supplied by the left anterior descending artery. The main part of the left bundle has dual supply from distal right coronary artery and proximal left coronary artery, the posterior division of the left bundle is supplied by distal portion of left circumflex artery.

ARRHYTHMIA:

The term arrhythmia generally refers to all rhythms other than regular sinus rhythm. Even slight variation caused by autonomic activity during respiratory cycle is termed as sinus arrhythmia. Literally arrhythmia means “imperfection in a regularly recurring motion”[23]

Arrhythmias are classified as bradyarrhythmias (rate<60/min) , tachyarrhythmias (> 100/ min). Arrhythmias may occur as primary or secondary disorder. Primary disorder reflects the basic or essential abnormality,classified into 1. Disturbance in impulse formation 2. Disturbance in impulse conduction- either block or re entry secondary disorder occur as a result of, or secondary to primary disorder, tertiary disorder reflect defect in conduction sequence.[24]

PRIMARY DISORDERS OF RHYTHM

Disturbance in impulse formation

1.Sinus rhythm :

- Sinus arrhythmia
- Sinus tachycardia
- Sinus bradycardia

2. Ectopic rhythm ;

- Atrial extrasystoles
- Paroxysmal atrial tachycardia
- Atrial fibrillation
- Atrial flutter

3. AV nodal rhythm :

- Av nodal extra systoles
- Extrasystolic -Paroxysmal - AV nodal tachycardia
- Idionodal tachycardia

4.Ventricular rhythm :

- Ventricular extra systoles
- Extra systolic ventricular tachycardia
- Idioventricular tachycardia
- Ventricular fibrillation
- Ventricular flutter

Disturbance in impulse conduction:

- Sino atrial block
- Atrioventricular block
- Reciprocal rhythm
- Re - Entrant tachycardia

SECONDARY DISORDERS OF RHYTHM

1.Escape rhythm:

- Atrial escape
- AV nodal escape
- Ventricular escape

2. AV dissociation

3. Phasic aberrant ventricular conduction

MECHANISM OF ARRHYTHMOGENESIS

TACHYARRHYTHMIA :

- **Abnormal automaicity :**

It maybe due to any one of the following mechanism,

1. Enhanced/ Ectopic pacemaker activity
2. Trigerred activity

- Early After Depolarization:

  – Delayed After Depolarization

  –

Enhancement / Ectopic pacemaker :

Normal or subsidiary pacemaker activity can be enhanced, leading to sinus tachycardia or a shift to ectopic sites within the atria, giving rise to atrial tachycardia. One cause can be enhanced sympathetic nerve activity. Another can be the flow of injury current between partially depolarized myocardium and normally polarized latent pacemaker cells. This mechanism is thought to be responsible for ectopic beats that occur at the borders of ischemic zones[25,26].Other causes of enhanced pacemaker activity include a decrease in the extracellular potassium levels as well as acute stretch. of the Purkinje system can occur in akinetic areas after acute ischemia[27] Accelerated idioventricular rhythms have been attributed to enhanced normal automaticity in the His-Purkinje system.[28]

Triggered activity:

Triggered activity is initiated by after-depolarization, which is a depolarizing oscillation in the membrane voltage induced by one or more preceding action potentials. Thus, triggered activity is pacemaker activity that results as a consequence of a preceding impulse or series of impulses, without which electrical quiescence would have occurred. This triggering activity is not caused by an automatic self-generating mechanism, and the term *triggered automaticity* is therefore contradictory. These depolarization can occur before or after full repolarization of the fiber and are best termed *early afterdepolarizations* (EADs), when they arise from a reduced level of membrane potential during phases 2 (type 1) and 3 (type 2) of the cardiac action

potential or are termed *late* or *delayed afterdepolarizations* (DADs), when they occur after completion of repolarization (phase 4), generally at a more negative membrane potential than that from which EADs arise.

Abnormal conduction:

Re - entry;

This is considered to be the most common mechanism of sustained paroxysmal tachyarrhythmia. Re entry is primarily due to abnormal mode of conduction or impulse may re circulate in a closed loop and cause repetitive activation without the need for new impulse. There are 5 types of re-entrant circuit models (figure-of-eight, spiral, leading crcuit, ring reentrant model) have explained. It could be either a functional or anatomic reentrant mechanism. Requirement for the reentry include :

1. Electrophysiologic inhomogenity: (differences in conduction and or refractionaries) in two or more region of heart connected with each other to form potential closed loop.
2. Unidirectional block in one pathway
3. Slow conduction in alternate pathway allowing time for the initially blocked pathway to recover
4. Re excitation of initially blocked pathway to complete loop activation.

The functional reentrant mechanisms can underlie many forms of tachyarrhythmias in ischemic or /infarcted hearts[29] . Comtois and colleagues recently concluded that the spiral waves concept better explains the functional re -entrant cardiac arrhythmias[30]. Figure of eihgt model can underlie ventricular arrhythmias originating from a thin surviving epicardial layer overlying infarction.Repetitive circulation of impulse over this loop produce sustained arrhythmia.

Block (Brady arrhythmia):

Suppression of automaticity of SAnode or impairment of conduction in any one component of the conductive system, either due to enhanced vagal activity or ischemic damage to the conductive tissues leads to Brady arrhythmias or block

ARRHYTHMOGENESIS IN THE INFARCT ZONE:

The incidence of arrhythmias is higher in patients, the earlier they are seen after the onset of symptoms. Many serious arrhythmias develop before hospitalization. Almost 90% of the patient develop rhythm abnormalities after acute myocardial infarction**,** 67% of Ventricular tachyarrhythmias occur with in 12 hrs of MI[31],[32] After acute infarction 25% of patients experience conduction disturbances within 24 hours.

Tachyarrhythmias during acute phase of myocardial infarction result from reperfusion, altered autonomic tone and/or hemodynamic instability. Non infarcted adjoining ischemic region is responsible for majority of arrhythmia[33]. The presence of injury current may directly enhance phase 4 depolarization of purkinje cells resulting in increased automaticity. Anaerobic metabolism of this region resulting from hypoxia

leads to acidosis with low levels of ATP. Spontaneous or therapeutic reperfusion results in increased production of oxygen free radical, intracellular Ca, increased catecholamine levels. An effect of oxidative stress on sodium channel function also postulated to play a role in post infarct arrhythmias. The electrophysiologic correlates of these cellular abnormalities include *slowing of conduction and prolongation of refractoriness, they provide ideal situation for re-entrant arrhythmias*. Increased catecholamine and calcium overload lead to *increased automaticity and enhanced after depolarization.* Arrhythmias in the remodeled ischemic myocardium is evolving from sites of slow conduction near the border zone, which is characterized by rate dependant slowing of re entry. Fibers stretch resulting from increased ventricular end diastolic pressure also arrhythmogenic.[33]

Bradyarrhythmias during the initial few hours of acute myocardial infarction, usually results from enhanced vagal activity, which are usually benign. But conduction disturbances beyond first 24 hours require most attention. It indicates necrotic damage of the conduction tissue, these conduction blocks will produce much complications.[32]

There are emerging clinical techniques to measure area at risk for arrhythmias. Measurement of certain parameters with MRI (surface area of the infarct and amount of ventricular mass damaged by the infarct) predict the incidence VT better than EF measured by echocardiogram[34]. The extent of peri-infarct zone measured by MRI is correlated with risk of arrhythmias.[35]

TACHYARRHYTHMIAS:

Sinus tachycardia;

About 30% of patients experience sinus tachycardia[36]. It usually represents an appropriate physiologic response to left ventricular dysfunction left ventricular dysfunction or stimulation of sympathetic nervous system. Management requires ACE inhibitors, diuretics for left ventricular dysfunction and beta blockers to reduce sympathetic activity.

Supraventricacular tachyarrhythmias:

Supraventricacular tachycardia such as atrial tachycardia,and atrio ventricular tachycardia have been reported to occur only in 1% of patients with acute myocardial infarction.

Atrial fibrillation ;

Atrial fibrillation is seen in 5-10 % of patients with acute myocardial infarction, most commonly in those who have significant left ventricular dysfunction and congestive heart failure[37]. The presence of atrial fibrillation in patients with myocardial infarction increases in-hospital, early, and late mortality after acute myocardial infarction. GUSTO III(Global Union of Strategies to Open occluded coronary arteries) reported 30 day mortality of 15% with odds ratio – 1.49 after adjusting for other post myocardial infarction complications before onset of atrial fibrillation.GUSTO III sub study showed that only class 1 antiarrhythmics, and sotalol were associated with better outcome and compared amiadarone or electrical cardioversion. Sinus rhythm was

restored in 70% of patients. But these drugs showed negative outcome in CAST [38](Cardiac Arrhythmia Suppression Trial), SWORD (Survival With Oral d Sotalol) trials [39]. The above two are long term studies which assessed the usefulness of class 1 drugs and d-sotalol in the presence of ventricular arrhythmia and sudden cardiac death. Another class III drug amiadarone is also very effective for atrial fibrillation in myocardial infarction as reported in EMIAT,[40] CAMIAT[41]. Persistence or recurrence of atrial fibrillation despite the administration above drugs is a major concern. In this situation rate control strategy with AV nodal blocking agents will be equally safe. Anticoagulation with warfarin should be started.

Atrial flutter ;

Atrial flutter is not commonly seen in patients with acute myocardial infarction. It has been reported to occur in only about 1% of patients. Treatment approach is similar to atrial fibrillation but long term therapy is usually not required.

Ventricular arrhythmias:

Ventricular arrhythmia may be ventricular premature complex (VPC) ,idioventricular rhythm,or ventricular tachycardia and ventricular fibrillation. 60% of ventricular arrhythmias occur within 12 hours of acute myocardial infarction. Sustained ventricular arrhythmias occur in upto 20% of patients with acute myocardial infarction[32]

Ventricular premature complexes:

This is the most common type of rhythm disturbance following acute myocardial infarction. In the past, frequent, multifocal , early diastolic ventricular extrasystoles and

R-on –T phenomenon were considered as warning arrhythmias for the development of ventricular fibrillation and ventricular tachycardia. But GISSI -2 reported that ventricular premature complexes of > 10 / hr predicted greater risk of malignant arrhythmia, sudden deaths in patients who did not receive thrombolytics. But in those who received thrombolytic therapy, ventricular premature complex did not predict sudden death due to arrhythmia, until the frequency is > 25/ hr[42,43]. It is now clear, however that such warning arrhythmias occur with same frequency in many patients whether subsequently develop VF or not. Infact several studies have demonstrated that primary VF occurs even without any antecedent warning arrhythmias, and patients can develop VF despite pharmacologic suppression warning arrhythmias. Based on this evidence, most clinicians pursue a conservative course. If VPC s encountered in the presence of sinus tachycardia early in the process of evolving MI, the use of iv beta blockers may reduce the incidence of subsequent VF.

Accelerated Idioventricular rhythm (AIVR) :

AIVR is seen in almost 20% of patients with AMI[44].It is defined as wide QRS complex with rate of 60—100 beats per minute. It results from either due to,

1. Failure of SA and AV node, as a result of structural damage or enhanced vagal tone mediated suppression of nodal automaticity, which allows the ventricular focus to escape

2. An abnormal ectopic focus with in the ventricles may assume the role of dominant pacemaker due to enhanced automaticity as a result of ischemia induced

reperfusion injury.

Most of the episodes of AIVR are of short duration and terminate spontaneously. It does not presage malignant arrhythmias. AIVR due to failure of proximal pacemaker represents the stable rhythm. However, AIVR due to an enhanced ectopic focus, it is potentially an unstable rhythm.If it is sustained and produce hemodynamic compromise, then atropine, or pacemaker may be required

Ventricular tachycardia ;

VT is defined as 3 more ventricular premature complexes occurring consecutively at a rate of >120/min . If it lasts for less than 30 sec, it is called as Non Sustained VT, if persists more than 30 seconds or causing hemodynamic compromise that requires intervention then it is labeled as SustainedVT. Based on the morphology of QRS, VT is classified as monomorphic or polymorphic ventricular tachycardia.

Monomorphic VT :

VT with uniform QRS morphology and fairly constant RR interval is defined as monomorphic ventricular tachycardia. When it occurs with in 2 days of AMI it is Early VT, and those occurring beyond 48hrs is Late VT. It has been reported to occur in 6.2%of patients with AMI as per GUSTO I trial. Incidence of VT has come down recently as reported in GUSTO III (4.7 %). MILLS ,GISS-I[45] trials have demonstrated that VT or VF during hospital stay, do not influence the one year mortality Finally GUSTO investigators concluded that early ventricular arrhythmias (<48 hrs)may not increase long term risk .But the incidence of late VT and VF negatively influence the

one year outcome. This difference is due to difference in the pathophysiology of early and late arrhythmias.

Polymorphic VT:

VT with multiple QRS morphology, varying amplitude and cycle length is defined as polymorphic ventricular tachycardia. It has been reported in 2% of patients with AMI, most of the time it rapidly becomes unstable and degenerate into ventricular fibrillation and produce hemodynamic compromise. [46]

.**SUSTAINED Vs Non –SUSTAINED VT:**

Non-sustained VT in the immediate peri-infarction period does not worsen the outcome. But the patients who experience multiple runs of non-sustained VT are having greater risk for hemodynamic compromise. Likewise the incidence of non-sustained VT in patients with depressed left ventricular function (EF<40%) or non-sustained VT beyond 48 hrs of AMI are considered as markers of sudden cardiac death[47]. In this subgroup electro physiologic testing and therapy is required. SustainedVT requires immediate termination with procainamide or amiodarone. Pulseless VT should be given DC shock of 200 J synchronized with QRS.

Ventricular fibrillation :

VF occurring with in 48 hrs of MI is defined as primary VF,incidence is greatest in the first hour following MI and rapidly declines thereafter. Approximately 60% occurs with in 4 hrs, 80% with in 12 hrs. Secondary or late VF which occurs after 48hrs of MI usually associated with pump failure, cardiogenic shock. Risk of secondary VF increase with large infarct size and anterior wall MI. Secondary VF in association with cardiogenic shock has poor prognosis with higher in-hospital mortality rate of about 40 -60% . Primary VF has uncertain prognostic implication.

BRADYARRHYTHMIAS

Sinus bradycardia :

Sinus bradycardia is defined as sinus rhythm with rate of < 60 per minute. This is a common arrhythmia in patients with inferior and posterior wall MI. It is present in approximately 40% of individuals .The postulated mechanism is stimulation of vagal afferent receptors which are more common in the infero-posterior portion of the left ventricle This may result in bradycardia and hypotension as a manifestation of Bezold Jarisch reflex.Vasovagal response to pain also produce bradycardia

.Atrio –Ventricular block

First degree AV block :

First degree AV block is defined as prolongation of PR nterval more than0.20 sec, it occurs approximately in 15%[48] of patients with AMI,most commonly in those having inferior wall MI. Almost all patients who develop 1st degree AV block have

conduction disturbance located above the level of bundle of His. This distinction is important, because the progression to complete heart block or ventricular asystole occurs most commonly in those with conduction disturbance below the bundle of His. Since in acute MI, the block is above the bundle of His, the clinical significance is usually minimal and no specific therapy is indicated. If first degree AV block is associated with severe bradycardia and hypotension, atropine has to be administered

Second degree AV block :

Second degree AV block manifest as intermittent failure of atrial impulse to activate the ventricles and exists in two forms

1. Mobitztype1 or Wenkebach AV block
2. Mobitz type II block

Mobitz Type I or Wenkebach AV block:

Mobitz type I block is observed in 6% of patients with AMI[49]. It is defined as progressive lengthening of PR interval with gradual shortening of RR interval followed by a drop in QRScomplex (ie) failure of conduction of the sinus impulse to reach the ventricles. It usually occurs due to ischemia of AV node, most commonly associated with inferior MI. Most of the time it is transient and does not affect the prognosis. Since the lower pacemaker is capable of maintaining the heart rate and cardiac output, it usually requires no treatment. If ventricular rate is unable to sustain perfusion, immediate treatment with atropine is required. In the thrombolytic era, Brazilian study showed drastic reduction in the incidence of Mobitz type 1 block (1.8%).

Mobitz Type II block:

Mobitz type II block is uncommon, it represents only 10% the second degree AV block and the overall incidence is about 1%. It is defined as an intermittent failure of conduction of the sinus impulse to reach ventricles with uniform PR interval of the conducted beats before the non conducted one. Mobitz type II block is characterized by 1.Conduction abnormality, located below the bundle of His 2.Usually has wide QRS complex 3. Mostly associated with anterior wall infarction 4.It often progress suddenly to complete heart bliock 5. It is associated with poor prognosis.The mortality rate associated with progression to complete heart block is approximately 80%. Mobitz type II block should be immediately treated with transcutaneous pacing or atropine

Third degree AV block :

Third degree AVblock or complete AV block has been reported to occur in 20% patients with AMI during pre thrombolytic era. In the thrombolytic era incidence has come down to 5-8%[50] .MILLS study developed scoring system to predict the occurrence of complete heart block. Prognosis with this condition depends on location of the block and the size of the infarct. In patients with inferior MI, usually the block occurs at or above the level of bundle of His (in about 70% of cases) and escape rhythm is usually stable with rate of more than 40/min. In the remaining cases the block is situated below the bundle of His and resulting in an escape rhythm with rate of < 40/min which may compromise the hemodynamic status. Complete heart block in most of the patients with inferior wall MI responds to pharmacologic intervention alone. The mortality rate is

about 15% - 40%. In patients with anterior MI usually the 3rd degree AV block is preceded by an intraventricular conduction block or Mobitz type II block in such cases the conduction disturbance usually located below the bundle of His. The rhythm may suddenly progress to asystole and associated with an in-hospital mortality rate of 60%-80%. Immediate treatment with atropine or transcutaneous pacing is required for this population. Patients with anterior MI who develop 3rd degree AV block and survive into hospitalization often require a permanent pacemaker.

Bundle Branch Block

Approximately 15% of patients with AMI develop block at one or more of the three fascicles. Isolated left anterior fascicular block (LAFB) occurs in upto 3 -5% and is unlikely to progress to complete heart block. Isolated posterior fascicular block (LPFB) occurs in about 1-2% [51]of patients .It is usually associated with larger infarct and results in higher mortality. Right bundle branch block occurs in about 5-10% of individuals, either as isolated one or in association with hemi block. RBBB is usually seen with infarct in the anteroseptal region. The combination of RBBB with LAFB or LPFB is called as bifascicular block. Bifascicular block with prolonged PR interval is termed as trifascicular block. Nearly 40% of such patients progress to complete heart block. Left bundle branch block is reported to occur in about 2-3.5% of patients with AMI. Among the patients with any BBB, the in-hospital mortality rate is about 29.7-32.5% [52] and the late mortality is around 12%. Occurrence of any BBB in the setting of acute MI increased the risk of mortality by 4.4 fold.

OUTCOME

Outcome in AMI can be assessed in three phases: in hospital, early (<_30day) and late (beyond 30 days) .

MORTALITY:

The overall mortality rate following STEMI is approximately 4-7% in the published clinical trials[9].However; this is not the case in real world situation, which can be obtained from registry data[11]. In the registry data from Europe, the mortality data is around three times higher than what has been observed in clinical trials .In a study published in Scotland, the case fatality rate is about 22.2% [53].In MITRA,MIR [54]registry data from Germany, the overall mortality is around 15% In India, CREATE registry data recorded 30 day mortality rate of about 8.6% in STEMI .Study from Vellore in South India, reported in hospital mortality of 16.9% in a group of 1320 patients with acute STEMI. In the past two decades mortality rate in AMI has decreased by 30%. Even then, one in 25 patients who survives initial hospitalization dies with in one year of AMI

MORBIDITY :

Among the spectrum of acute coronary syndromes patients with STEMI are having the maximum number of complications because of transmural involvement. Other infarct related factors responsible are larger size of infarct and absence of tissue perfusion at the microvacular level [55,56]

Complications ;

1. Disturbance in the electrical activity – Arrhythmias

2. Myocardial dysfunction - pump failure

3. Mechanical disruption of cardiac structures –VSR,MR

PUMP FAILURE :

The clinical syndrome of pump failure in AMI results chiefly from systolic (contractile) and diastolic dysfunction. In some patients, it may result from other complications such as acute mitral regurgitation, left to right shunt as a result of septal rupture, cardiac tamponade from free wall rupture or right ventricular dysfunction. It may be worsened or precipitated by additional factors like sustained or recurrent supraventricular or ventricular arrhythmias, relative or absolute hypovolemia and negative ionotropic drugs[57.] Basic pathology responsible for this complication is ventricular remodelling

PATHOPHYSIOLOGY OF PUMP FAILURE

Remodelling :

Remodelling of myocardium refers to structural and functional changes in the infarct zone and in remote normal myocardium., which begins with in minutes after acute MI and continues for month or years .

Changes include:

1 Infarct expansion

2. Dilatation and hypertrophy of remaining normal myocardium

3. Interstitial fibrosis with resultant impairment of contraction and relaxation

4. Global change in the shape of left ventricle from its normal elongated ellipse to spherical[58]

Infarct expansion ;

An increase in the size of the infarcted segment known as *infarct expansion*, is defined as "acute dilation and thinning of the area of infarction not explained by additional myocardial necrosis."[59] Basic pathophysiology behind this is defective infarct healing and /or increased left ventricle stress with slippage of necrotic myofibrils with resultant thinning of infarct zone. It occurs in about 35-42% of patients with transmural anterior or anteroapical infarction. This process can be reversible if coronary flow is reestablished rapidly before structural changes occur. However it may progress, if the flow is not reestablished or established late after the occurrence of structural changes.[60] Absence myocardial reperfusion at microcirculatory level even in the presence of TIMI III flow may produce progressive LV dilatation.Infarct expansion leads to complications like pump failure, LV aneurysm, mitral regurgitation, mural thrombus emboli and myocardial rupture.

Contactile (syslolic) dysfunction ;

Four abnormal patterns of contractile dysfunction develop in sequence following acute interruption of blood flow (1) dyssynergy (2)regional hypokinesia (reduced contraction) (3)akinesia (lack of contraction) (4)dyskinesia (paradoxic systolic bulging) [61] Infarct expansion and aneurysmal dilatation will reduce mechanical efficiency of LV (wasted work) as a result of paradoxical bulging .Progressive abnormalities of regional

myocardial function result in decreased stroke volume and increased LV end systolic volume. Ultimate end result will be reduced Ejection Fraction. In general with involvement of 10% of LV mass reduction in EF will be negligible. However, involvement >40% LV mass usually results in fatal cardiogenic shock.

Diastolic dysfunction :

Diastolic dysfunction with reduced LVcompliance is relatively a common cause of pulmonary congestion in AMI. .It is due to alteration in viscoelastic properities of ischemic and necrotic tissue as a result of cellular and interstitial edema during acute phase, healing with fibrosis in subacute and chronic phase .Reperfusion into the areas of irreversible necrosis leads to the formation of contraction band necrosis and resulting in acutely stiff infarct zone.[62]Acute RV dilation in right ventricular MI also contributes to diastolic dysfunction of LV through leftward bulging of interventricular septum. (Burnheim effect) These diastolic abnormalities impede left ventricular filling with resultant rise in the left ventricular filling pressure and decrease in the stroke volume even with good LV systolic function. Co-morbid conditions like diabetes mellitus, systemic hypertension increase the vulnerability for diastolic dysfunction

CARDIAC RUPTURE :

Mechanical disruption of intracardiac structures may occur through the zone of necrosis in the left ventricular free wall, septum, papillary muscle or contiguous chordae tendinae. Cardiac rupture of any form may contribute roughly about 15% of all fatalities of acute MI, free wall rupture alone is accounting for 85%. Reperfusion therapy has

reduced the overall incidence of cardiac rupture[63,64]. Incidence of papillary muscle rupture has been reported in 39% of patients, septal rupture in 0.2-0.4% and the free wall rupture in 1-2%[65,66]patients with AMI . Failure to reestablish flow even at microvasculature level is a risk factor for myocardial rupture.

Left ventricular failure:

Killip clinical classification system stratifies patients based on clinical evidence of left ventricular failure [67], as in the following table.

Killip class	Clinical features	Hemodynamic status	Mortality
Class I (No heart failure)		CI > 2.2 PCWP <18mmof Hg	1-3%
Class II (Mild heart failure)	Basal rales +/-- S3 gallop	CI > 2.2 PCWP <18mmof Hg	3-5%
Class III (Pulmonary edema)	Dyspnoea,S3 gallop Pulmonary rales	CI > 2.2 PCWP <18mmof Hg	5-25%
Cass IV Cardigenic shock		CI < 2.2 PCWP <18mmof Hg CI < 2.2 PCWP >18mmof Hg	30-60%

Congestive heart failure occurs in approximately 15-20% of patients with acute MI. NRMI-2 study reported the incidence of heart failure in 19% of cases. Killip class II

in13.6%, class III in 5.6% of patients. Incidence of death is 21.4%in patients with heart failure which is significantly higher than patients without heart failure. 7.2%[68].

CARDIOGENIC SHOCK :

This is the leading cause of death in AMI. The criteria for Cardiogenic shock consist of [69]

1. Hypotension with systolic BP <90mmof Hg for atleast 30min which requires vasopressor or IABP support

2. Clinical evidence of end organ hypo perfusion like oliguria

3. Confirmatory radiographic evidence - pulmonary congestion

hemodynamic features –PCWP>15mmof Hg .CI <2.2 lit /min /m ^{2}

Incidence of cardiogenic shock is nearly 8% in large registries[70].LV failure is the leading cause responsible for 79% of cardiogenic shock in patients with AMI. Other causes include acute severe MR (6.5%), VSR (3.9%), isolated RV shock (2.8%) and others (6.7%). Mortality rate in patients with cardiogenic shock following acute MI is 60%[71]. In older studies it was around 80-90%.[72]

AIM OF THE STUDY

1. To estimate the incidence of various types of arrhythmias in patients with acute ST elevation myocardial infarction .

2. To analyze the pattern of arrhythmias in relation to the different regions of the ventricle wall with ST elevation myocardial infarction.

3. To assess the in-hospital outcome, 30 day mortality in Arrhythmic and Non- arrhythmic population.

4. To analyze the influence of arrhythmias on the in- hospital outcome by comparing the outcome among the patients with and without arrhythmias.

MATERIALS AND METHODS

Three hundred patients admitted to the intensive coronary care unit of our Coimbatore medical college hospital between January 2007 and August 2008 with acute ST elevation myocardial infarction (STEMI) were studied in a prospective manner.

Acute STEMI was diagnosed according to the following criteria

Definition of STEMI:

1. Presence of chest pain of >20min duration

and

2. ST segment elevation of >1mm in atleast two standard limb leads

or >2mm in atleast two contiguous precordial leads or new onset of

Left bundle Branch block

and / or

3. CK- MB elevation.

STUDY PARTICIPANTS:

Inclusion criteria:

Patients who were presented within 12 hrs of onset of symptoms with evidence of STEMI and received thrombolytic therapy with streptokinase .

.**Exclusion criteria :**

1. Patients with Non STEMI or Unstable angina

2. People with previous history of coronary artery disease

3. People with previous history of arrhythmias

4. People with previous history of cardiomyopathy or heart failure

Patients who fulfilled the above inclusion criteria and not having any of the above said exclusion criteria were included in the study as a participant.

DEFINITION OF RISK FACTORS:

1. Smokers : Patients who currently smoke more than >10 cigarettes per day for longer than six months period were considered as smokers.

2. Hypertensives : Patients who were diagnosed to have hypertension as per Joint National committee 7(JNC-7) criteria [73] with or without end organ damage irrespective of their treatment status are considered as hypertensives,

3. Diabetes mellitus : Patients were considered as diabetic if their two random blood sugar were higher than 200mg/dl and later confirmed with fasting blood sugar value of more than 126 mg/dl or patients who were on treatment for diabetes mellitus

METHODS

12 lead ECG was taken for all patients, Leads V3R, V4R was taken in patients with inferior wall myocardial infarction Location of the infarct were defined as follows [74].

Regional wall of MI	Lead with ST elevation
Anteroseptal MI	V1-V3
Lateral Wall	V4-V6

High lateral	I ,aVL
Anterolateral MI	V1 –V6
Extensive anterior	V1 –V6 + I ,aVL
Inferior wall	II, III, avF
Inferior + Right ventricular MI	II, III, avF + V3R,V4R
Inferior + RVMI +Posterior	II,III, aVF + V3R,V4R V2 –Depressed and upward ST segment, wide tall Rwave , widened tall upright T wave

The data regarding the baseline characters of the patients like age, gender, smoking habit, diabetes mellitus and hypertension were recorded. Hemodynamic status of the patient were assessed by recording the pulse , blood pressur, jugular venous pressure (JVP). Careful auscultation of cardiovascular, respiratory system was done to look for the presence of S3, S4 gallop, murmur and crepitations, findings were recorded. Bloodsugar, urea, creatinine and electrolytes were done for all the patients at the time of admission.Chest X-ray, lipid profile has taken for all the patients before discharge..

All the patients were treated with antiplatelet drugs , sorbitrate morphine, atorvastatin ,ACE inhibitors, betablockers and with according to AHA /ACC guidelines as and when required .All the participants were put on continuous electrocardiographic monitoring for 24 hrs.12 lead ECG was repeated one hour after thrombolysis , every 24 hrs ,and also whenever the situation demanded .. Hemodynamic status assessed at regular intervals by clinical methods. Incidence of arrhythmias recognized promptly with continuous ECG monitoring and confirmed with 12 lead ECG most of the time. Stable patients transferred to the medical ward .Ejection Fraction, regional wall movement analysed

with echocardiogram. After discharge the patients were followed up in the outpatient department weekly for 30 days. Morbidity, mortality data were recorded .Prognostic significance of arrhythmia is analysed by comparing the outcome among the patients who developed arrhythmia and those who did not have any arrhythmia during the hospital stay.

Type of arrhythmia is categorized as per following definition[75]

Sinus tachycardia;

Regular narrow complex tachycardia with heart rate more than 100 /min with normal P-QRS -T relation.

Supraventricular tachycardia :

Regular tachycardia with rate of >125-200/min having narrow QRS complex, with absence of P wave or P wave which present as pseudo Q, pseudo R, pseudo S wave, or P wave distorting ST segment is defined as supraventricular tachycardia

Premature Atrial Contraction :

P wave occurring before the expected time with slightly different morphology, or negative Pwave occurring in a is similar manner in II III aVF called as premature atrial complex .This may or may not have conducted to ventricle. If it gets conducted it will be followed by narrow

QRS complex.

Paroxysmal Atrial Tachycardia (PAT) :

Regular narrow QRS tachycardia with abnormal Pwave morphology is defined as paroxysmal atrial tachycardia . According to the P, QRS ratio it can be labeled as to have 1:1, 2:1or 3:2 AVconduction. If the ratio is >1:1 the patient is said to have PAT with AV block.

Atrial flutter :

Atrial flutter is the expression rapid regular atrial excitation with atrial rate around 250-350/min characterized by regular, uniform, sharp, saw tooth like waves (F waves).The ventricular response is usually narrow QRS complex, regularity may vary depending on AV conduction

Atrial Fibrillation :

It is an irregularly occurring narrow QRS tachycardia with chaotic atrial activity producing fibrillatory P waves called as f waves. Atrial rate is usually >350/min. All the above supraventricular arrhythmias may have wide QRS if the conduction is occurring through a preexisting BBB or through an aberrant pathway .

Ventricular Prematue complexes :

Ventricular premature complexes are wide bizarre QRS complex >0.140 S which occur prior to expected next p wave. The pause following

VPC is fully compensated

Ventricular tachycardia ;

VT is defined as occurrence >_ 3 ventricular premature complexes in succession with a rate of >120/min . If it lasts for less than 30 sec it is non-sustained VT , if persists more than 30 seconds or causing hemodynamic compromise that requires intervention labeled as sustained VT. Based on the morphology of QRS , VT is classified as polymorphic or monomorphic ventricular tachycardia.

Ventricular fibrillation :

It is defined as irregular undulation of varying contour and amplitude on the ECG with absence of distinct QRS and T waves and prompt hemodynamic compromise requiring DC version

RBBB:

The diagnosis of RBBB is made when

1. Lead V1 reflects tall wide frequently notched R' deflection
2. Lead V5, V6 or I shows delayed widened S wave with increased QRS duration of 0.14 sec or longer

LBBB : The diagnosis of LBBB is made when

1. Lead V5, V6,I and aVL reflects a tall wide frequently

notched R' deflection

2. Lead V1 shows delayed widened S wave

with increased QRS duration of 0.14 sec or longer

Left Anterior fascicular Block :

1. Presence of left axis deviation > -- 60 degrees
2. Small Qin leads I and aVL with a small 'r' wave in II, III and aVF
3. Usually with normal QRS duration

Left Posterior Fascicular Block :

1. Right axis deviation > +120 degrees
2. Small "r" in leads I and aVL, with a small "q" II ,III and aVF
3. Usually with normal QRS duration
4. No evidence of Right ventricular hypertrophy

Bifascicular Block :

Presence of RBBB+LAFB with an axis abnormality which is not explainable by pathologic Q waves is labeled as left anterior hemiblock.. RBBB+LPFB in the absence of lateral wall infarction ,right ventricular hypertrophy or history of symptomatic chronic lung disease or corpulmonale is defined as Left posterior hemiblock

Trifascicular block :

RBBB + anyone type of fascicular block with prolonged PR interval of more than 0.12 s defined as trifasicular block

AVblock :

First degree AV block :

First degree AV block is defined as prolonged PR of more than 0.12 s with properly maintained P-QRS-T relation throughout .

Second degree AV block :

Type I ;

Mobitz type I block is said to be present if there is progressive lengthening of PR interval and shortening of RR interval followed by non conducted atrial activity (Pwaves) .

Type II ;

It is defined as an intermittent failure of the atrial impulse (Pwave) to be conducted to the ventricles with an uniform PR interval in all the conducted beats.

Third degree AV block :

Third degree AV block is said to be present if complete AV dissociation is present with ventricular rate less than 60 /min

High degree AV block :

This is defined as Type II 2nd degree AV block or 3rd degree AV block in which 3 or more P waves left unconducted and this may develop following Type I 2nd degree block or without recognized 2nd degree block.

Killip Class :

Class I - No heart failure

Class II - mild heart failure - presence of S3 gallop ,basal crepitations

Class III - Pulmonary edema – presence of S3 gallop Pulmonary rales, rontgenographic evidence of pulmonary edema

Class IV - Cardiogenic shock

1. hypotension with systolic BP<90mmof Hg for atleast 30mts and require vasopeessor or IABPsupport
2. clinical evidence of end organ hypoperfusion like oliguria (<20 ml /hr)

3.confirmatory radiographic -pulmonary congestion

STASTICAL ANALYSIS :

All the data were analyzed with SPSS software (version13.0) . Categorical variables were compared by Chi square test(X^2 test) or Fischer exact test and continuous variables were presented as mean +/-

SD and were compared by Student “t” test. A probability value of <0.05 was considered statistically significant.

RESULTS AND ANALYSIS

Three hundred patients with acute ST Elevation MI were analyzed . Clinical and demographic characteristics are summarized in Table 1. Among the study population anterior wall MI was seen in 177 (59%) patients .In this group 59 (19.6%) were having Anteroseptal, 62 (20.7%) were anterolateral MI, and 56 (18.7%) had extensive anterior wall MI. Inferior wall involvement was present in 111 (37%)patients. Of whom 65,(21.7%) had isolated Inferiorwall MI, 23(7.7%) had Inferior +Right ventricle involvement, 17 (5.7%) were having Inferior+RV+Posterior wall MI and Inferior +posterior involvement was present in6 (2%) patients. High lateral and lateral wall MI includes 9 (3%) patients , Anterior+Inferior wall MI in 3(1%) patients

Table 1 : Baseline characters of study population

Totally 132 (44%) patients have experienced significant arrhythmias The most common type of arrhythmia noticed was VPC, almost in 71% of participants.

Analysis of Risk Factors :

Among 177 patients with anterior wall MI, 84 developed significant arrhythmias. In the inferior wall group, 48out of 123 have developed arrhythmias(47.5% Vs 39.0% P value <0.05 Relative risk 1.2) .So the association between anterior wall MI and incidence of arrhythmia is significant .Moreover anterior wall MI increase the risk of arrhythmia by 1.2 fold than inferior wall MI. Out of 132 patients with arrhythmias 113 were males (85.6%) compared to non arrhythmic population where 123/168 were males(85.6%Vs 73.2% P value <0.01) . Likewise 93 patients among arrhythmic group (n= 132) were smokers, in non-arrhythmic group (n=162) 98 were smokers (70.5% Vs 58.3% Pvalue < 0.05). Seventy four patients in arrhythmic group were having diabetes mellitus, among non-arrhythmic 68 out of 162 were diabetics (56%Vs 40.5% pvalue < 0.05). Sixty one (46.2%) arrhythmic patients were hypertensives compared to 35.7% among non-arrhythmic P value < 0.05. Table 3 shows the distribution of risk factors in patients with and

without arrhythmia.

Fig. 1 Arrhythmias in relation to the Regional Wall

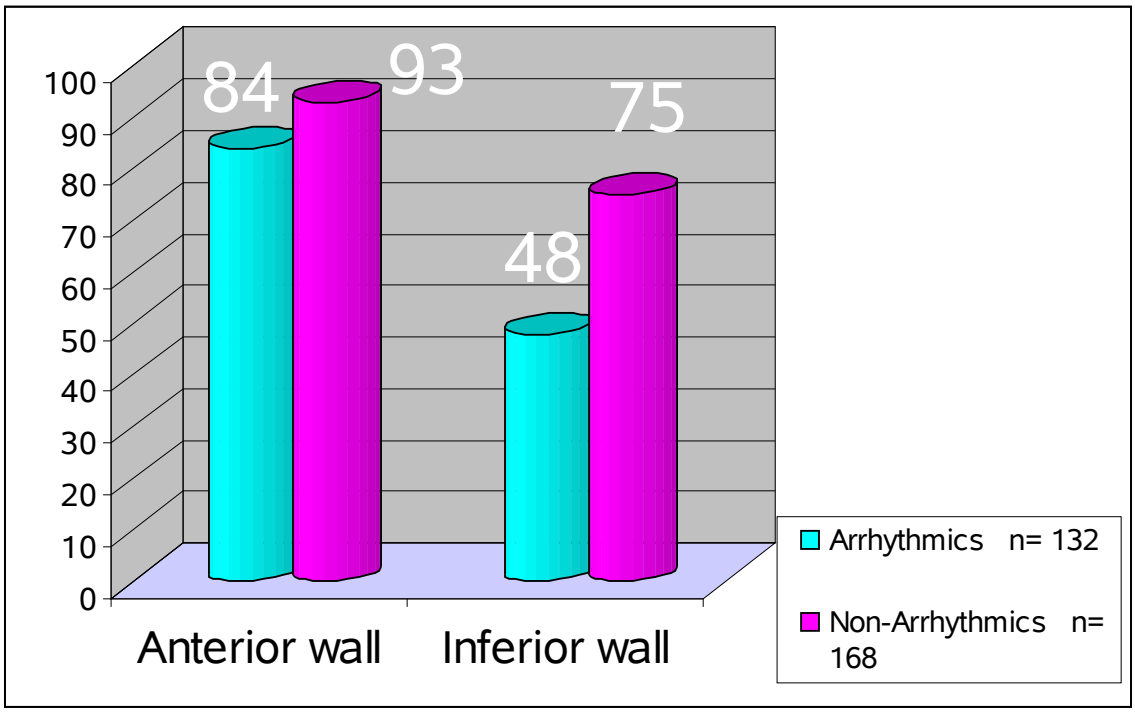


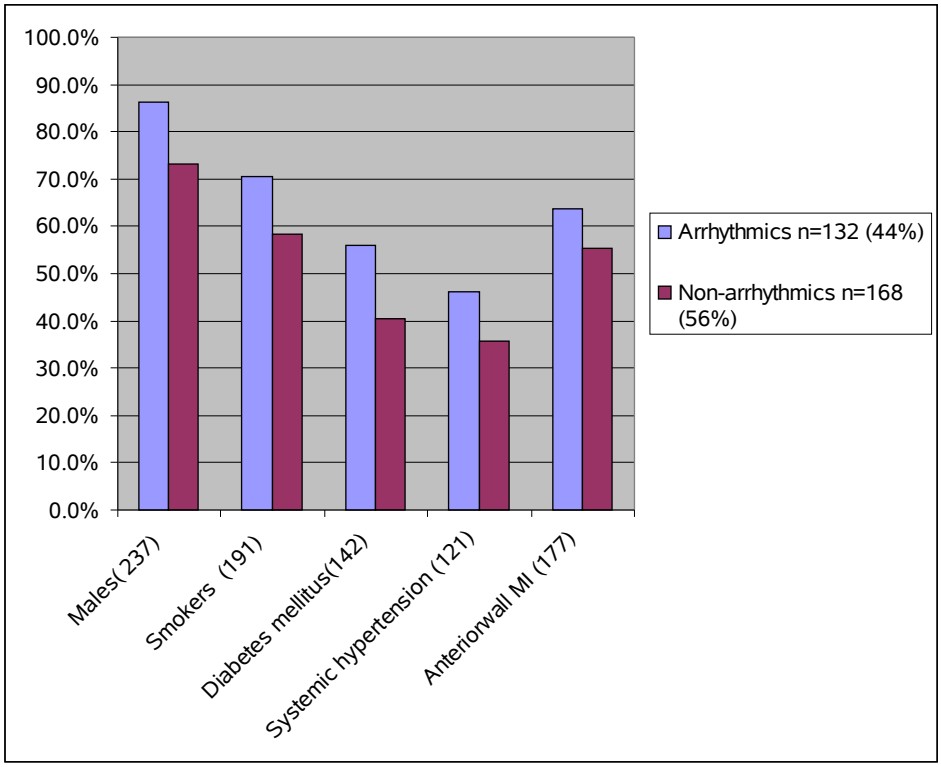
Table 2

Relative Risk = 1.2

Table 3 : Risk factor analysis

Baseline Characters of Study Population						
Characters		Arrhythmics (n= 132)		Non-Arrhythmics (n=168)		
		Count (n)	Column N %	Count (n)	Column N %	P value
Age	(Mean +/--S.D)	45 +/-- 7		51 +/-- 8		*
Sex	Female	18	13.6%	45	26.8%	*
	Male	114	86.4%	123	73.2%	< 0.05
	smokers	93	70.5%	98	58.3%	< 0.01
	Diabetics	74	56.1%	68	40.5%	< 0.03
	Hypertensives	61	46.2%	60	35.7%	< 0.05
	Anteriorwall MI	84	63.6%	93	55.4%	NS
	Inferiorwall MI	48	36.4%	75	44.6%	NS
Ejection Fraction	(Mean +/--S.D)	43 +/-11		48 +/-11		< 0.05

Fig . 2 RISK FACTORS OF ARRHYTHMIA



The above analysis shows that anterior wallMI, male sex along with diabetes mellitus, smoking and hypertension are important risk factors for the incidence arrhythmia.

Incidence of arrhythmias

Among 300 study population 132 had significant arrhymias. Of whom 70 (53 %) patients had tachyarrhythmia62 (47 %) patients had bradyarrhythmia. Among the tachyarrhythmias 35 (50%) were ventricular arrhythmias(VT=17(24.3%),VF=18(24.7 %)) , 35 (50%) were

supraventricular arrhythmias (Atrialfibrillation=30 (42.5%) Atrialflutter=2(2.9%), Supraventricular tachycardia=3 (4.3%)) . Fiftyone of 70 (72.9%) tachyarrhythmia have occurred in patients with anterior wall MI , the remaining 19(27.1%) have occurred in inferior wall and others.In the bradyarrhythmia group 33of 62 (53.2%) were Bundle branch block (LBBB = 9 (14.5%), RBBB = 13 (21%), LAFB = 4 (6.5%) LPFB = 1 (1.6%), BiFB =4(6.5%), TriFB= 2 (3.2%)), 29of 62 (46.8%) were AVblock (2^{nd} degree type1 = 4 (6.5%), type 2= 2 (3.2%), 3^{rd} degree (CHB) =23 (37.1%)). Of 62 bradyarrhythmias 33(53.2%) were in anterior wall 29 (46.8%) were in inferior wall MI

The incidence of different types of arrhythmias among entire study population (n=300) is shown in Figure.3 & Table.4 . Figures 4&5 are showing the frequencies of different types of arrhythmias among arrhythmic population(n=132) in relation with regional wall of MI .

Fig 3

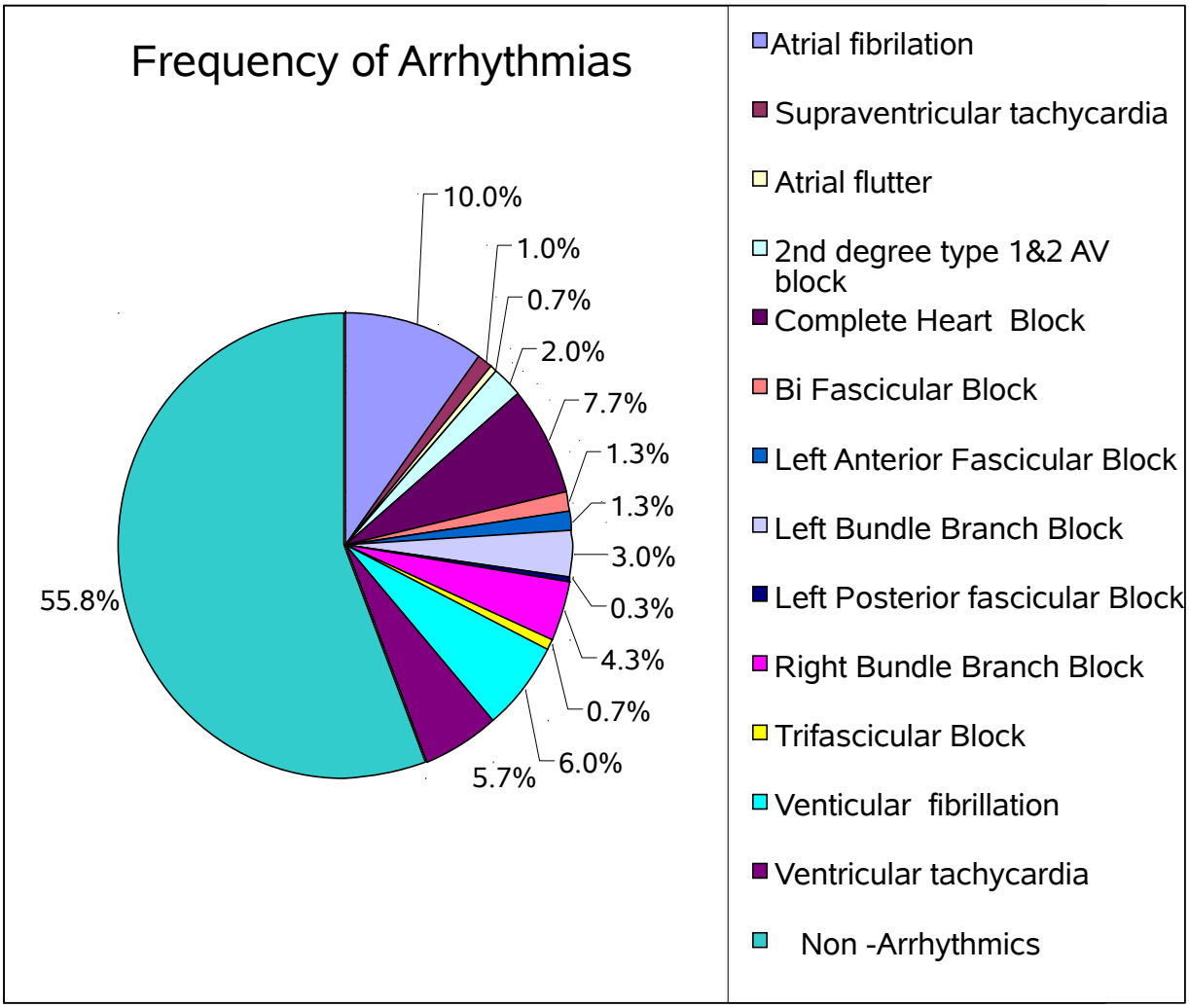


Table 4

Incidence in relation to Regional Wall of MI

In the anterior wall MI group (n=177) ,84 (63.6%) have developed arrhythmias out of them 51(60.7%) were having tachyarrhythmias ,33 (39.3%) had bradyarhythmias . Atrialfibrillation is

the most common type 17out of 84 (20.2%) followed by VF=15 (17.9.%)VT = 14(16.7 %) .Among the patients with inferior wall MI (123) , 48 (39%) have developed arrhythmias , Bradyarrhythmia is more common among inferior wall MI (n=29, 60.4%) than tachyarrhythmias (n= 19 , 39.6%) (pvalue <0.01) .Complete heart block is the most common type n=18 (37.1%),

Fig4: Types of Arrhythmia in anterior wall of MI

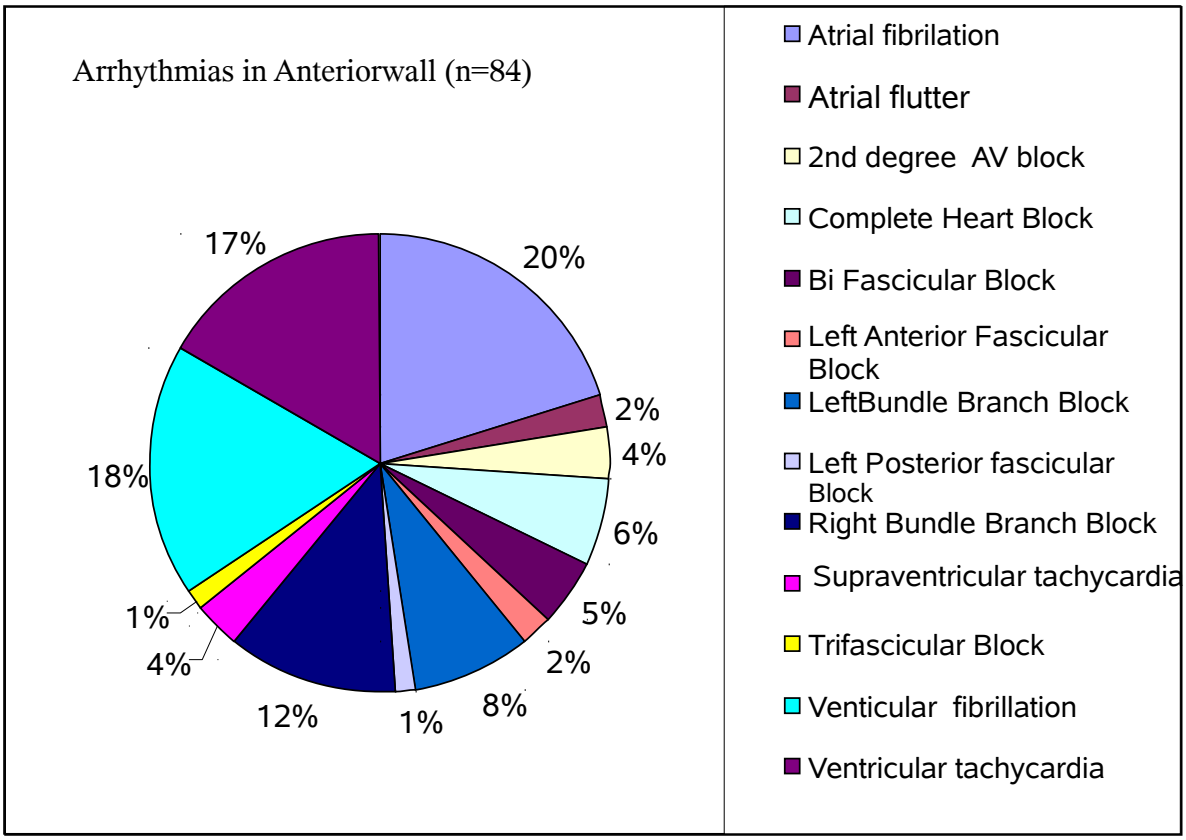


Fig5: Types of Arrhythmia in inferior wall MI

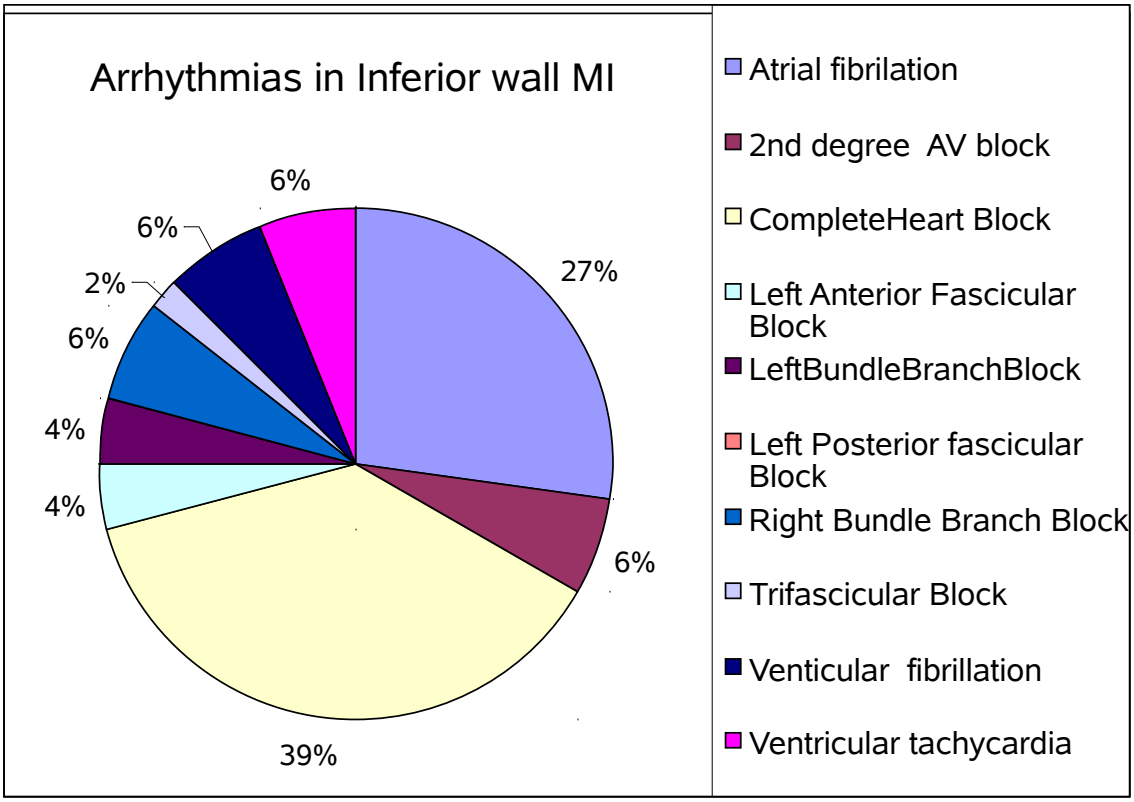
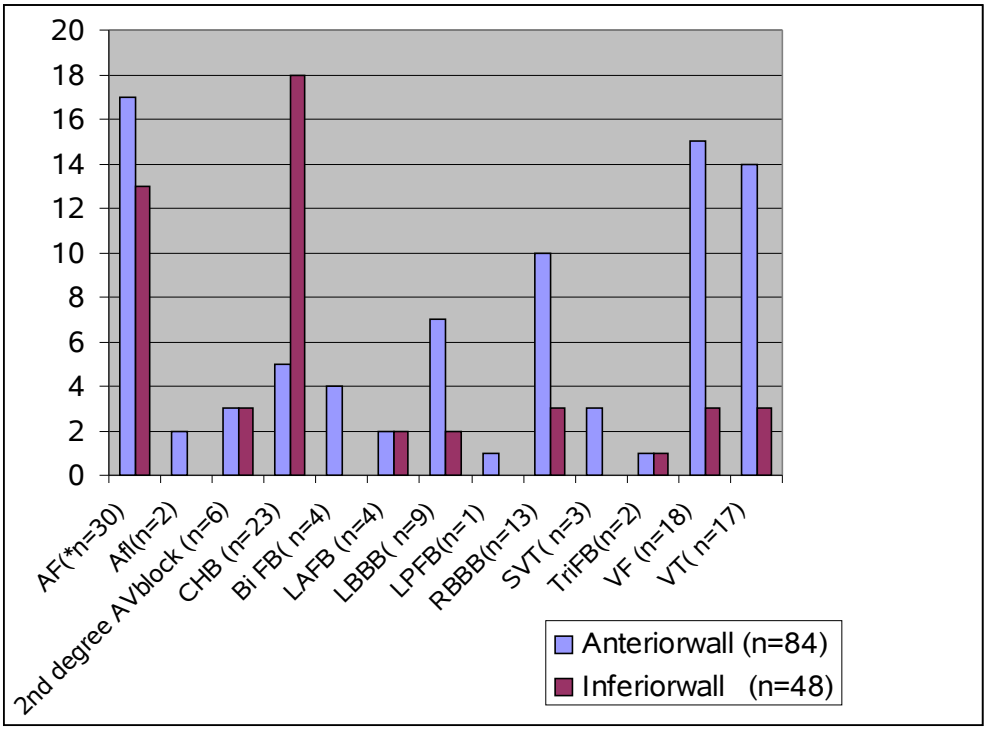


Fig. 6: Comparison of incidence of arrhythmia in relation to regional wall of ventricle with MI



ANALYSIS OF OUTCOME

HOSPITAL EVENTS:

Outcome data during the period of hospital stay and during the next 30 day period also observed . Out of 300patients 53 (17.7%) were died during the hospital stay,70 (23.3%) patients had cardiac failure (Killip class II ,III) and 39(13%)had cardiogenic shock.

Table.5 summarizes in-hospital events among arrhythmic and non – arrhythmic population. Patients with arrhythmia had higher incidence cardiacfailure 49out of 132 (37.1%) compared to21 out of168 in non arrhythmics (P value <0.01 relative risk =3). Incidence of arrhythmia,increase the risk of cardiogenic shock by 2.9 fold .Twenty seven of 132(20.5%) arrhythmics had cardiogenic shock compared to non-arrhythmic group where 12out of 168(7.1%)experienced shock, Pvalue <0.01.Likewise arrhythmic population were at increased risk of dying. Mortality in arrhythmic group = 39 /132 (29.5%) significantly higher than non arrhythmic group 14/ (8.3%) Pvalue <0.01 (RR 3.5)

Table.5 : **Hospital events in Arrhythmic & Non- Arrhythmic population**

HOSPITAL EVENTS		
	Ar rhythmics	Non- Arrhythmics (n=168)

	(n= 132)				
	Count n=132	(%)	Count n=168	(%)	P Value
Ejection Fraction (Mean +/-- S.D)	43 +/-- 11		48 +/-- 11		< 0.01
Killip class1	78	59 %	138	82.1 %	< 0.01
Killip class 2	33	25 %	14	8.3 %	< 0.01
Killip class 3	16	12.1%	7	4.2 %	< 0.01
Hge	0	0.0	1	0.6 %	*
MR	3	2.3 %	2	1.2 %	*
VSR	1	0.8 %	0	0.0	*
CardiogenicShock	27	20.5 %	12	7.1 %	< 0.01
Mortality	39	29.5 %	14	8.3 %	< 0.01

Fig 7 : Major events among study groups

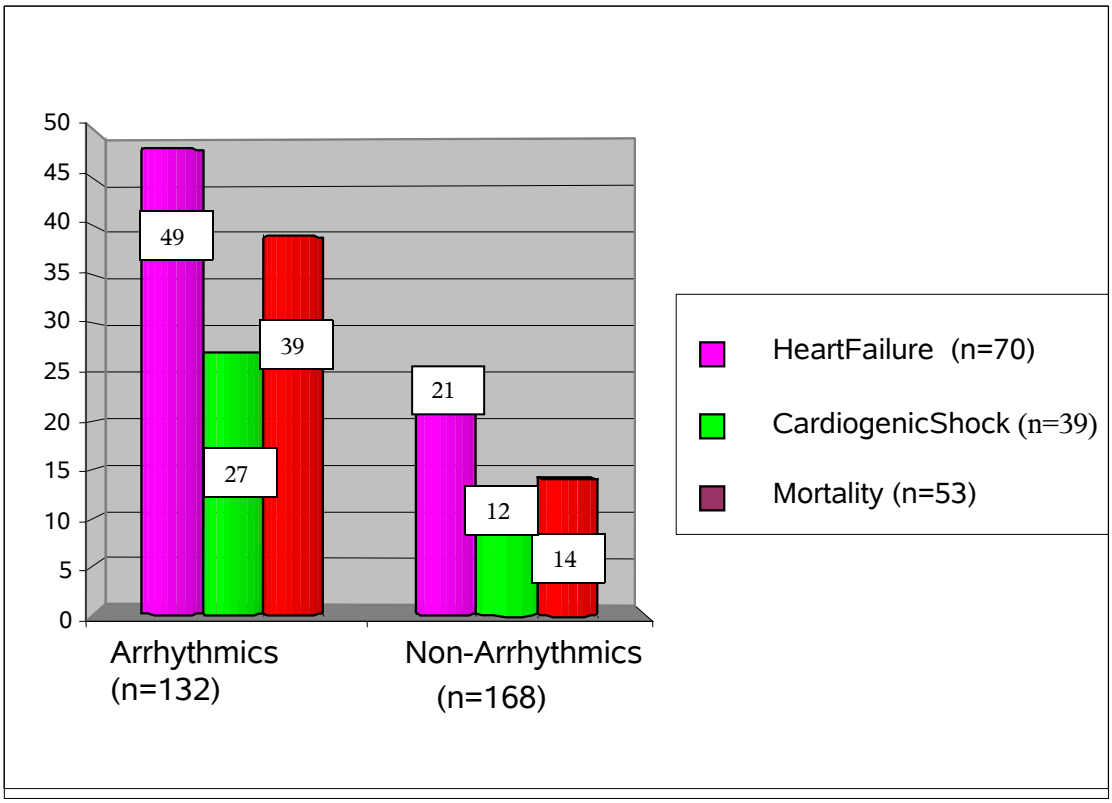
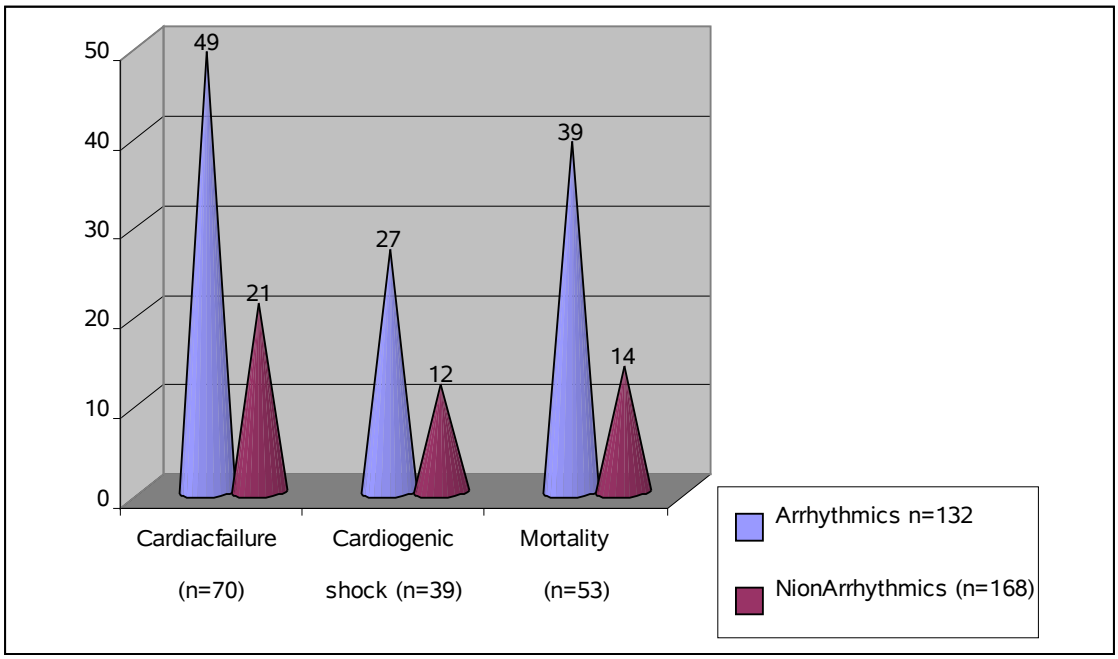


Table:6 Analysis of Major events in arrhythmic & non –arrhythmic population

Fig 8: Outcome in Arrhythmic & Non- Arrhythmic population – Comparison



OUTCOME IN RELATION TO TYPE OF ARRHYTHMIA

Supraventricular arrhythmias ;

Atrial fibrillation is observed as the most common type of supraventricular arrhythmia in this study 30/35 patients(85.7%). Others were SVT 3cases (8.6.%),atrialflutter in 2 (5.7%). patients. AF was responsible for the entire hospital events in the supraventricular arrhythmia group . Nine patients (25.7%)developed cardiac failure , 7 (20%) patients had cardiogenic shock and 6 patients died (17.1%). Patients who has anterior wall MI with atrial fibrillation had poor outcome, mortality rate is 30%, incidence of cardiac failure is 55.6%.

Incidence of atrial fibrillation significantly reduces the ejection fraction (EF %), 43+/--11 % compared to 48+/-11 % in non-arrhythmic population

Ventricular arrhythmias

Ventricular fibrillation and ventricular tachycardia are the significant members in this group ,Others like VPC ,accelerated idovemntricular rhythm did not affect outcome. Totally 35 patients had significant ventricular arrhythmias (VF = 18 , VT =17) . In patients with VF mortality was 13 (72.2%) , incidence of cardiac failure and shock were 61.1% ,27.8% respectively Among patients with VT incidence of mortality was 52.9%, cardiac failure, shock were 70.6%, and 29.4%. VT ,VF in patients with anterolateral MI resulted in poor outcome than in other location , mortality rate is 70%, 78% respectively .Ejection fraction in anterior ,anterolateral MI is 39% significantly lower than in other location.

Bundle Branch Block

The observed incidence of any one type of intraventricular coduction defect (isolated RBBB, LBBB ,Fascicular Bifascicular ,or trifascicular block) was 11% (n=33) In this group left bunldle (9patients) and right bundle branch block (n=13)are the common types .Cardiac failure occurred 11 patients (33.3%) four patients had shock (12.1%) mortality rate was 18.2% (n=6). Patients with isolated RBBB had 7.7% mortality LBBB had 22.2% mortality rate. The patients with bifascicular block ,trifascicular block had a mortality rate of 25%, 50% respectively. One patient had left posterior fascicular block, he died following acute pulmonary edema and shock. Mean EF among patients with bundle branch block was 43+/--9 %

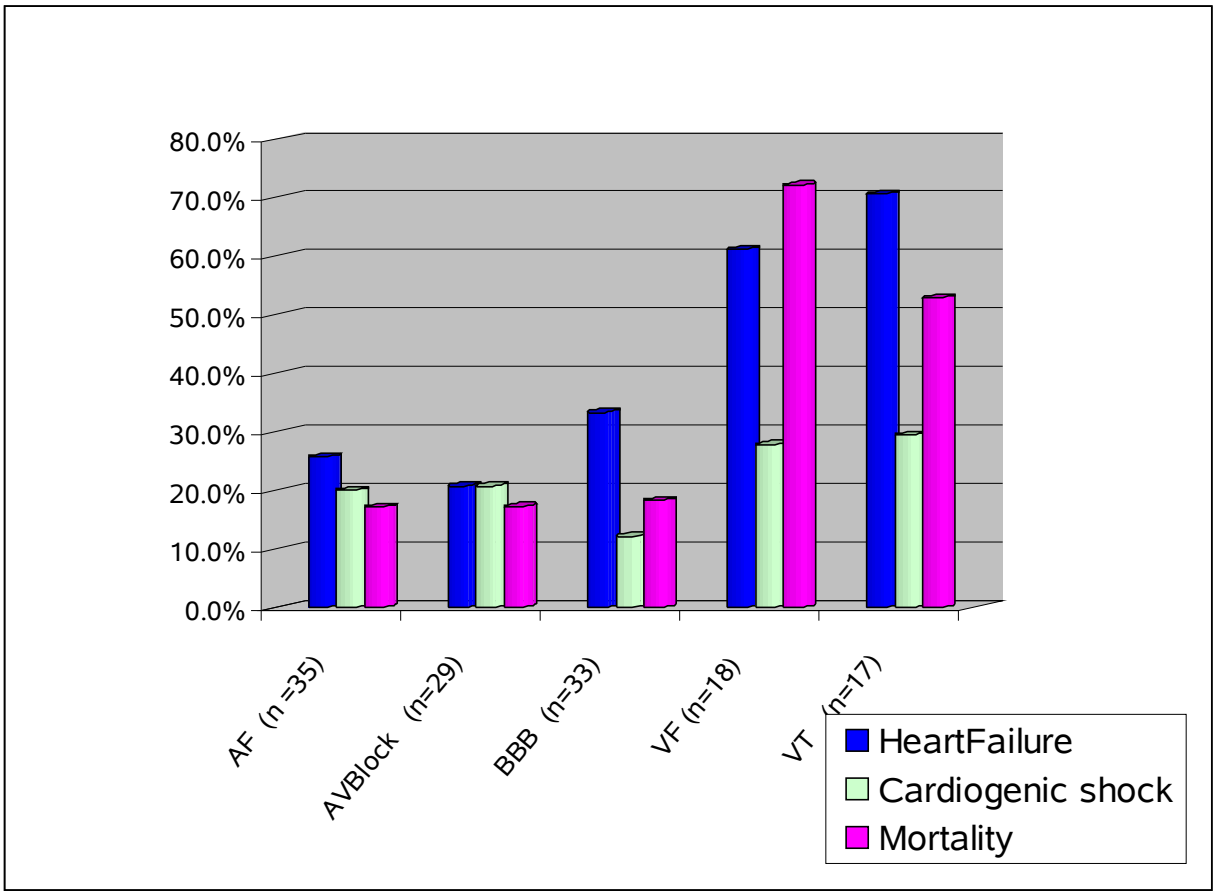
Atriventricular block

Totally 29 patients had AV block ,(9.7%) ,6 (2%)of them had 2nd degree AV block (2nd degree type I=3 ,type II=3) 23 (7.6%) had 3rd degree AVblock .In 2nd degree AV block mortality rate was 16.6% ,in which all 3 patients with 2nd degree type I block are alive did not have any complications .In 2nd degree type II block (n=3) one patient died, mortality rate was 33.3%, Among patients with complete heart block(n=23) 4 patients died, mortality rate was (17.4%), Overall

mortality rate in patients with AV block was (17.2%) and 6 (20.6%) patients had cardiac failure, 6 (20.6%) patients had cardiogenic shock .Incidence of complete heart block in anterior wall MI resulted higher mortality (50%) than other types of AV block.

Table 7 : In hospital outcome in different groups of Arrhythmia

Fig 9 Comparison of outcome among different groups of arrhythmia



Ejection Fraction

Mean EF% in patients with supraventricular arrhythmias, BBB , VT ,are 43+/--11 , 43+/--9 38+/--6 respectively .but in non arrhythmic population is 48+/-- 11 Pvalue <0.05 Patients with AV block had mean EF of 44+/11 Pvalue was not significant .Difference between EF among patients with VF and without arrhythmia was not significant But notable point here is that the echocardiogram has done only for 5of 18patients remaining 13 were died , so EF can not be interpreted Patients with anterior wall MI and arrhythmia had lower EF (41%) than patients with

inferiorwall MI and arrhythmia (48%)

Cardiac failure

Seventy of 300patients (23.3%) developed cardiac failure. Among those 47 (67.1%) were Killip class II and 23 (32.9%) were Killip class III . Ejection fraction (EF) in Killip I was 48+/--10 %, in class II ,III were 37+/--10% ,34+/--6% (Pvalue <0.05). Out of 70 patients with cardiac failure 49 (70%) (P<0.001)had any one type of significant arrhythmia preceding the event. Out of these 49 patients with arrhythmia and heart fuilre 23 (46.9%) progressed to cardiogenic shock, 33 (67.3%) patients were died during the hospital stay. Patients with anterior wall MI and arrhythmia (n = 84) had higher incidence of hearfailure38 (45.2%) compared to those with inferiorwall MI and arrhythmia is the (23%) (P<0.01) .

Cardiogenic shock

Cardiogenic shock occurred in 39 (13%) patients .Twenty seven (69.2%)of them had preceding arrhythmia. Cardiac failure is the commonest cause of shock, n=30 (76.9%) others are mitral regurgitation in 3 patients(7.6%) ventricular septal rupture in one patient (2.5%) ,right ventricular infarction (RVMI) in 5(12.8%). Mortality rate was (71.8%) among all patients with shock irrespective of arrhythmia status.

Mortality

The overall in hospital mortality rate was 17.7% (n=53). Of whom39 (73.6%) patients were having arrhythmia 14 (26.4%) form non arrhythmic group .When we consider individual type, patients with ventricular fibrillation were having higher rate of mortality (13/18) 72.2%. **Table 8** : Hospital events – contribution from different type of Arrhythmias

Fig10 – Heart Failure – contribution from different type of Arrhythmias

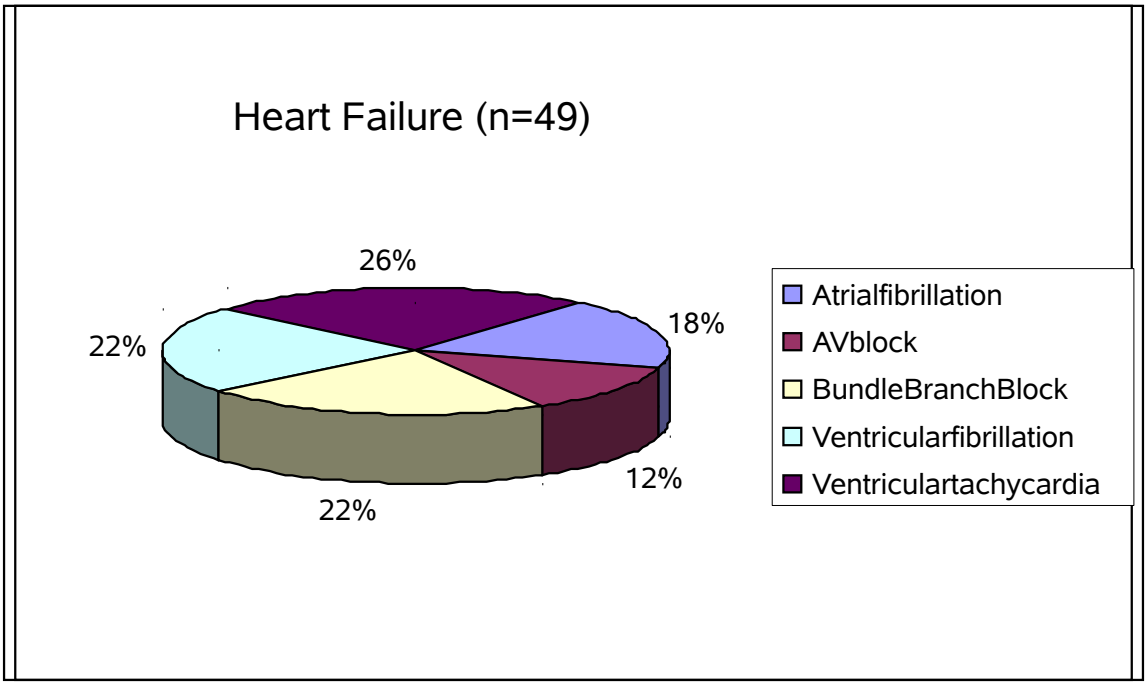


Fig 11 – Cardiogenic shock –contribution from different type of Arrhythmias

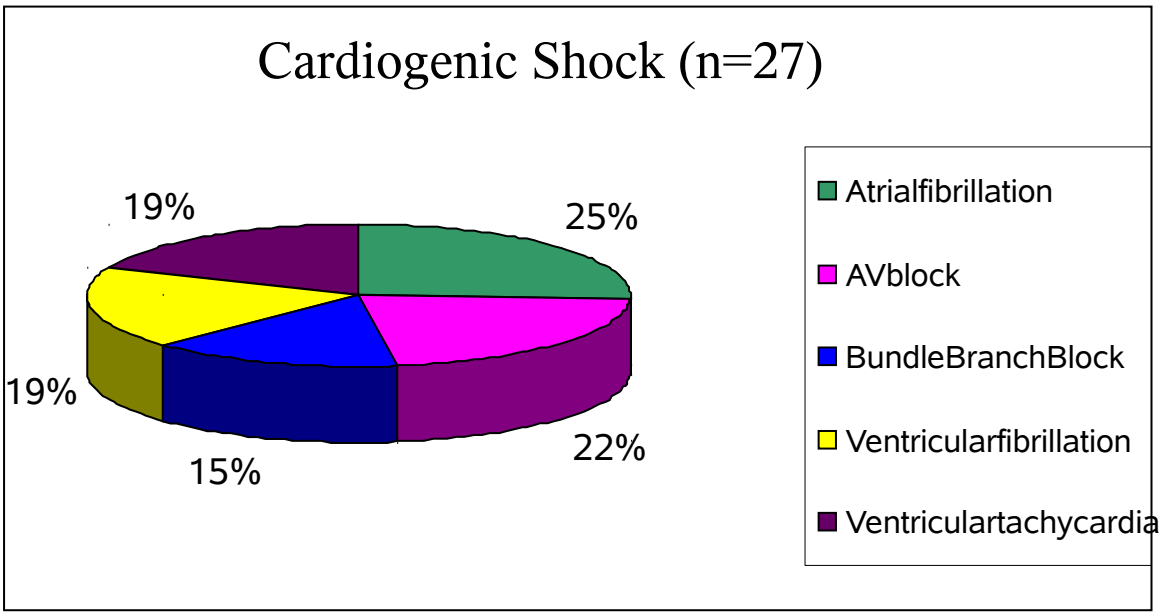
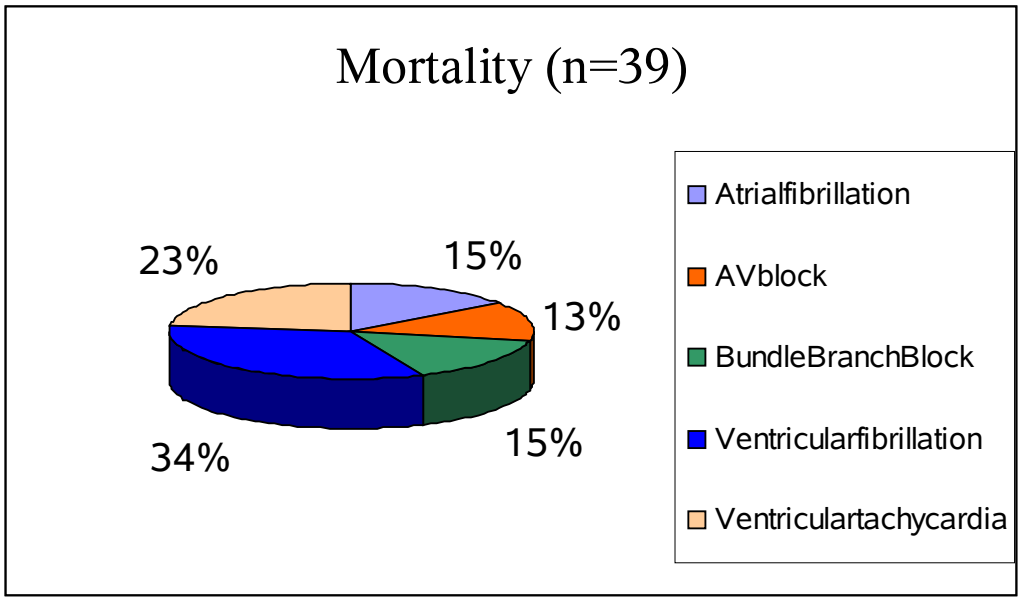


Fig 12 - Mortality –contribution from different type of

Arrhythmias



30day follow up

30 day follow up data was available for 200 patients. 15 (7.5%) patients developed heart failure. Three (1.5%) patients died during this period. One patient presented with recurrence of VT reverted with pharmacological treatment

DISCUSSION

Incidence of arrhythmias ,

In our study 300 patients were analyzed. Ventricular premature complex is the most common type observed with incidence rate of 67% most during the intial hours after thrombolysis. This findng is similar to previous done by Morrison et al(70%)Arthur Moss (57%).

Supra ventricular arrhythmias

Incidence of SVT in our study 1%. Atrial flutter observed in two patients (0.7%). Similar to other reports these two supra ventricular arrhythmias did not have any major hospital events had benign course. Among the incidence of supra ventricular arrhythmias atrial fibrillation tops the list (10%) Incidence is similar to other studies during thrombolytic era[76, 77] . AF was transient in 30% did not require therapy. Out of remaining (21 patients) 7 patients received cardioversion in addition to amiadarone others received amiadarone only .Sinus rhythm restored in 70% at the time of discharge .Of the 30 patients with AF, 30% developed heart failure, 23.3% had cardiogenic shock 20% died during the hospital stay. Mortality rate and hospital events were higher compared to GUSTO III[78, 79] .The possible reason could be the use of sotalol, and class I antiarrhythmics in GUSTO III study which

demonstrated that the later drugs provide better in hospital outcome than amiadarone and cardioversion in new onset AF after acute MI .

Ventricular arrhythmias:

In our series the incidence of ventricular tachycardia, ventricular fibrillation were 5.7%, 6% repectively which is slightly higher than the incidence in GUSTOIII[80], (4.7%, 4.8%) GISSI-2[81] (3.9%)but similar to GUSTO I [82] (6.2%, 6.7%) In our observation when considering VT ,VF together the incidence of cardiac failure and shock were 65.7%, 28.5% respectively, the mortality rate was 62.8% .Eventhough the cardiac failure and shock are more common among VT patients, the mortality rate was lower than patients with VF But overall the hospital events were higher in our population compared to GUSTO I ,III (34.6%of cardiac failure, 20% of shock 44% of inhospital mortality).This may be due to higher number of patients in our study had co-morbid condition like diabetes mellitus, systemic hypertension , high door to needle time which is mainly due to unawareness of symptoms .

Atrioventricular block :

We found that the incidence of AV block was 9.7%. Second degree AV block has occurred in 2%, complete AVblock in 7.7% of patients with acute MI. This finding is similar to GUSTO [83]study report, which

observed incidence about 9.1% in patients treated with streptokinase but higher than the GISSI-2[84] report (5.5%) Harpez et al observation[85], and a Brazilian study[51] report (4.7%). Majority of complete AV block (72.4%) occurred in patients with inferior wall MI with or without right ventricular involvement. Mortality rate in patients with inferior wall MI and AV block was 9.5%. In the anterior wall group it was 37.5% . Overall mortality rate in patients with AV block was 17.2% This observation is much lower than other studies which reported 35..4%. This difference may be due transient nature of AV block, majority of AV block in our study have occurred among inferior wall group. In 45% of patients AV block recovered with in 72 hrs did not produce much hemodynamic compromise

Bundle Branch Block :

Bundle branch block of any type either isolated or bifascicular ,trifascicular had occurred in 11% of study population . Isolated RBBB occurred in 13 (4.3%) patients, LBBB occurred in 3%(n=9) of patients. Melgarejo-Moreno et al[86] in a multicenter study of 1238 consecutive cases acute MI described 10.7% frequency of isolated RBBB or associated with hemiblock. He described incidence of LBBB in 3.3% cases in an another study.[87] Newby et al [88] in 681 AMI patients described

incidence of any RBBB of 13% and LBBB in 7% patients using Holter monitor. This higher incidence may be due to the use of Holter monitoring. Brazilian study reported incidence of 4.5 % RBBB, 2.3% cases of LBBB. In our study 33.3% of patients with BBB developed cardiac failure 12.1% developed shock. Mortality rate was 18..2%. This observation is lower than the incidence in Brazilian study (32.4%) But similar to report by Alan S et al [89]

Table 9

BBB	Our study 2008)		Alan S et al (1998)		Brazilian study (2002)	
	Incidence	Mortality	Incidence	Mortality	Incidence	Mortality
RBBB	4.3%	7.6%	6.7%	20.3%	3%	21.4%
LBBB	3%	22.2%	6.2%	23.6%	2.3%	38.1%

Out come - Influence of arrhythmia

The overall in hospital mortality was 17.7% in our study .In the randomized trials of STEMI patients the reported mortality ranged from 4 to7% . A Study from Vellore in South India reported in-hospital mortality of about 16.9% . Mortality rate among patients with and without arrhythmia were 29.5% ,8.3% respectively .This shows that occurrence of arrhythmia during acute STEMI increase the mortality rate by 3.5 times than those without arrhythmia. Incidence of any arrhythmia after

acute STEMI resulted in 3 fold increase in the risk of heart failure 2.9 fold increase in the risk for cardiogenic shock. The higher mortality in our patients may be due to the following reasons, Firstly all of our patients received thrombolytic therapy alone with streptokinase no percutaneous interventions was being done. Average Door to needle time in our study was about 5 hrs because of ignorance of the symptom, transportation delay. So most of our patients have lost the “golden hour”. Available treatment options for arrhythmia in our hospital were amiadarone and cardioversion other new drugs like ibutelide , procainamide, sotalol were not available .For the management of cardiac failure hemodynamic monitoring and IABP were not possible , The observations of our study may be taken as reflection of real Indian situation with limited resource .

CONCLUSION

1. 44% of patients with STEMI had anyone type of clinically significant arrhythmia.patients with anterior wall STEMI had higher incidence of arrhythmia(n=84 of 177) (47.5%)than STEMI in other location(39%)
2. Tachyarrhythmia is more common than bradyarrhythmia accounts for 53% (n=70)of arrhythmic population (n=132)
3. Atrialfibrillation is the most frequent type of with incidence rate of 10%(n=30) followed by complete heart block 7.6% (n=23) ,VF ,6% (n=18) VT 5.7% (n=17). 13 (4.3%)patients had RBBB. nine had LBBB(3%)
4. Male sex, diabetes mellitus, smoking ,systemic hypertension anterior wall involvement were the important riskfactors significantly associated with incidence of arrhythmia
5. Occurance of arrhythmia during acute STEMI increase the risk of mortality by 3.5fold, heart failure by 3fold ,cardiogenic shock by 2.9 fold, and significantly reduces the ejection fraction (EF%
6. In hospital events were higher among patients with tachyarrhythmias.

than bradyarrhythmias. The observed mortality rate was 60%

(n=42of 70patients) .Ventricular tachycardia and fibrillation were the major contributors .

7. The mortality rate among patients with bradyarrhythmia was 17.7% .patients with LBBB had higher mortality(22.2%) than RBBB(7.6%) .
8. In addition to diabetes mellitus, smoking , anterior wall MI, incidence of arrhythmia after acute STEMI is an independent predictor of poor prognosis.
9. Event rate during 30day follow up was (7.5%) heart failure ,1.5% mortality (3of 200 patients)
10. Incidence of arrhythmia and its complication in patients with STEMI still remains high in resource poor settings Early recognition and treatment with appropriate drug, electrical cardioversion may improve the outcome .

BIBILOGRAPHY

1 Lopez AD, Mathers CD, Ezatti M, et al .Global and regional burden of disease and risk factors2001: systematic analysis of population health data, Lancet 2006;367:1747-57

2 Reddy KS, Cardiovascular disease in non -Western countries NEngl J Med2004;350(24):2438-40

3 Ghaffer A, Reddy KS, Singhi M. Burden of non- communicable diseases in South Asia BMJ 2004; 328:807-10

4 Mohan V , Deepa R, Rani SS, Premlatha G. prevalence coronary artery disease and its relationship to lipids in selected populationin South India .The Chennai Urban Population Study (CUPS No 5) J Am coll Cardiol2001;38:682-87

5 Joshi P, Islam S,PaisP,etal. Risk factors for early myocardial infarction in South Asians compared with individuals in other countries JAMA 2007;297:286-9

6 Fuster V, Badimon L, Badimon JJ, Chesebro JH. The

pathogenesis of coronary artery disease and the acute coronary syndromes. Part 1. *N Engl J Med* 1992:326;242–250.

7 Fuster V, Moreno PR, Fayad ZA, Corti R, Badimon JS. Atherothrombosis and high-risk plaque: part 1: evolving concepts. *J Am Coll Cardiol* 2005;46:937–954.

8 Pais P, Xavier D, Gupta R, et al .Treatment and outcome of acute coronary syndrome in India the (CREATE):a prospective analysis of registry data Lancet2008;371:1435-42

9 9 Global Utilization of Streptokinase & Tissue plasminogen activator for Occluded coronary arteries (GUSTO III) investigators. A comparison of reteplase with alteplase for acute myocardial infarction N Engl J Med 1997;337: 1118-23

10Assessment of the Safety and Efficacy of a New Thrombolytic (ASSENT-2) investigators. Single bolus tenacteplus compared with front loaded alteplase in acute myocardial infarction Lancet 1999;354:716-22

11. Brown N, Young T, Gray D etal. Inpatient deaths from acute myocardial infarction 1982-1992: analysis of data in

Nottingham heart attack register BMJ1997;315:159-164

10

11

12 Every NR, Freiderick PD, Robinson M, et al .A comparison of the National registry of myocardial infarction -2 with the co-operative cardiovascular project *J Am Coll Cardiol* 1999;33:1886-94

13 ZeymerU , Senges J. Why do we need prospective registries in patients with myocardial infarction Eu heart J 2003;24:1611-12

14 V Jacob Jose ,Satya N Gupta et al Morbidity and mortality of acute ST segment elevation myocardial infarction in the current era Indian Heart J2004;56:210-14

15 Hurst ' s .The Heart , Text book of cardiology 12th edition by Fuster, O'Rourke ,Walsh Chapter : 57

16

Pathologic basis of disease 7th edition 2004 by Robbins and Cotran chapter 12; page 573

17 Goldberg RJ, Spencer FA, Yarzebski J, et al. A 25-year perspective into the changing landscape of patients hospitalized with acute myocardial infarction (the Worcester Heart Attack Study). *Am J Cardiol* 2004;94(11):1373–1378. [PMID: 15566906]

18 Boersma E, Mercado N, Poldermans D, et al: Acute myocardial infarction. *Lancet* 2003; 361:847.

19 Ho SY, McCarthy KP, Ansari A, et al. Anatomy of the atrioventricular node and atrioventricular conduction system. *J Bifurcation & Chaos.* 2003;12:3665-3674

20 Josephson IR. Initiation and propagation of the cardiac action potential. In: Sperelakis N, Banks RO, eds. *Essentials of Physiology.* 2nd ed. Boston: Little, Brown, and Company, 1996;247–257.

21 Huser J, Blatter LA, Lipsius SL. Intracellular Ca^{2+} release contributes to automaticity in cat atrial pacemaker cells. *J Physiol.* 2000;524(pt 2):415.

22 Libby: Braunwald ‘s Heart disease ;A text book of cardiovascular medicine 8th edition chapter:31 p733-35

23 Galen S.Wagner Marriot’s Practical electrocardiography 2008 Eleventh edition chapter 12 ,p240-241

24 LeoShamroth :An introduction to Electrocardiography 7th edition 1990 Reprint 2005 Chapter:28 p321-26

25

Katzung BG, Hondeghem LM, Grant AO. Letter: cardiac ventricular automaticity induced by current of injury. *Pflugers Arch.* 1975;360:193. [PMID: 1237872]

26

Janse MJ, Van Capelle FJL. Electrotonic interactions across an inexcitable region as a cause of ectopic activity in acute regional myocardial ischemia. A study in intact porcine and canine hearts and computer models. *Circ Res.* 1982;50:527. [PMID: 7067060]

27 Vermeulen JT. Mechanisms of arrhythmias in heart failure. *J Cardiovasc Electrophysiol.* 1998;9:208. [PMID: 9511895]

28 Katz LN, Pick A. *Clinical Electrocardiography. Part 1. The Arrhythmias.* Philadelphia, PA: Lea and Febiger;1956:224, 225

29 Wit AL, Janse MJ. *The Ventricular Arrhythmias of Ischemia and Infarction. Electrophysiological Mechanisms.* Mount Kisco, NY: Futura; 1993.

30 Comtois P, Kneller J, Nattel S. Of circles and spirals: bridging the gap between the leading circle and spiral wave concepts of cardiac reentry. *Europace.* 2005;7(suppl 2):10.

31 Campbell RW, Murray A, Julian DG. Ventricular arrhythmias in first 12 hours of acute myocardial infarction. Natural history study. *Br Heart J* 1981;46(4):351–357. [PMID: 7295429]

32 Perron AD, Sweeney T. Arrhythmic complications of acute coronary syndromes. [PMID: 16199338) *Emerg Med Clin*

*North Am*2005;23(4):1065–1082.

33 Corr PB, Sobel BE. Mechanisms contributing to dysrhythmias induced by ischemia and their therapeutic implications. *Adv Cardiol* 1978;22:110–129. [PMID: 22992]

34 Bello D, Fieno DS, Kim RJ, et al. Infarct morphology identifies patients with substrate for sustained ventricular tachycardia. *J Am Coll Cardiol* 2005;45(7):1104–1108. [PMID: 15808771]

35 Yan AT, Shayne AJ, Brown KA, et al. Characterization of the peri-infarct zone by contrast-enhanced cardiac magnetic resonance imaging is a powerful predictor of post-myocardial infarction mortality. *Circulation* 2006;114(1):32–39. [PMID: 16801462]

Jewitt DE,Balcon R, Rafery,et al .Incidence and Manage ment of supraventricular arrhythmaisafteer acute myocardial infarction Am heart J 1969;77(2):290-293

36

37 Bhatia GS, Lip GY. Atrial fibrillation post-myocardial infarction: frequency, consequences, and management. *Curr Heart Fail Rep* 2004;1(4):149–155. [PMID: 16036038]

38 Echt DS, Liebson PR, Mitchell LB, et al. Mortality and morbidity in patients receiving encainide, flecainide, or placebo. The Cardiac Arrhythmia Suppression Trial. (CAST) *N Engl J Med* 1991;324(12):781–788. [PMID: 1900101]

39 Waldo A, Camm A, deRyuter H, et al. For the survival with oral d-sotalol(SWORD) investigators: effect of d-sotalol on mortality in patients with left ventricular dysfunction after recent and remote myocardial infarction. *Lancet* 1996;348:7–12. [PMID: 8691967]

40 Julian DG, Camm AJ, Frangin G, et al. Randomised trial of effect of amiodarone on mortality in patients with left-ventricular dysfunction after recent myocardial infarction: EMIAT. European Myocardial Infarct Amiodarone Trial Investigators. *Lancet* 1997;349(9053):667–674.

[PMID: 9078197]

41 Cairns JA, Connolly SJ, Roberts R, Gent M. Randomised trial of outcome after myocardial infarction in patients with frequent or repetitive ventricular premature depolarisations: CAMIAT. Canadian Amiodarone Myocardial Infarction Arrhythmia Trial Investigators. *Lancet* 1997;349(9053):675–682. [PMID: 9078198]

42 Statters DJ, Malik M, Redwood S, et al. Use of ventricular premature complexes for risk stratification after acute myocardial infarction in the thrombolytic era. *Am J Cardiol* 1996;77:133–138. [PMID: 8546079]

43 Zuanetti G, Nelson JM; Lantini R, et al, on behalf of GISSI-2 Investigators. Prognostic significance of heart rate variability in postmyocardial infarction patients in the fibrinolytic era: the GISSI-2 results. *Circulation* 1996;94:432â€“436. [PMID: 8759085]

44 Antman EM, Anbe DT, Armstrong PW, et al: ACC/AHA guidelines for the management of patients with ST-elevation myocardial infarction: A report of the American College of

Cardiology/American Heart Association Task Force on Practice Guidelines (Committee to Revise the 1999 Guidelines for the Management of Patients with Acute Myocardial Infarction(www.acc.org/clinical/guidelines/stemi/index.pdf).

45 Volpi A, Cavalli A, Santaro L, et al(GISSI Investigators) one year prognosis of early primary ventricular fibrillation in acute myocardial infarction:. *Am J Cardiol* 1989;63:1174-78]

46 Bigger JT Prevalence ,characteristics and significance of ventricualar tachycardia detected by 24 hour continuous electro cardiographic recordincs in the late phase of myocardial infarction Am J cardiol 1986:58(13):1151-60

47 Buxton AE, Lee K, DiCarlo L, et al. Nonsustained ventricular tachycardia in coronary artery disease: relation to inducible sustained ventricular tachycardia.
Ann Intern Med 1996;125:35–39. [PMID: 8644986]

48 Andrew D , Perron MD ,Timothy Sweeny, MD Arrhythmic complications of acute coronary syndrome .
Emerg Med Clin N Am 2005;23:1065-82

49 Hindman MC ,Wagner GS JaRo M et al The clinical significance of Bundle branch block complicating acute myocardial infarction Circulation 1978;58:679-688

50 Rathore SS, Gersh BJ, Berger PB, et al. Acute myocardial infarction complicated by heart block in the elderly: Prevalence and outcomes. *Am Heart J* 2001;141:47–54. [PMID: 11136486]

51 Claudia Camniha Escosteguy,Marcianode AlmeidaCarvalho,Robertode AndradeMedronho et al. Bundle branch block and Atrioventricular block as complications of acute myocardial infarctions in thrombolytic era. Arq Bras cardiol 2001; volume 76 (no4) :, 291-6

52 Alpman A ,Gudal M, Erol C,,et al The role of arrhythmia and left ventricular function in patients with acute myocardial infarction and bundle branch block Jpn Heart J. 1993;34(2):145-57

53 Capwell S Livingston BM ,Maclntyre Ketal Trends of case fatality rate in 117718 patients admitted with acute myocardial infarction in Scotland Eur Heart J 2000 ;21:1833-1840

54 Gottwik M, Zahn R, Schiele Ret al Differences in treatment and out come in patients with acute myocardial infarction with compared to deparment without cardiology results from pooled data of the Maximal Individual Therapy in Acute myocardial infarction(MITRA 1+2) registries and the Myocardial Infarction Registry (MIR) Eur Heart J 2001;22:1794-1801

55 Ito H Maruyama A Iwakura ket al Clinical implications of the no reflow phenomenon:a predictor of complications and left ventricular remodeling in reperfused anterior wall myocardial infarction Circulation;1996; 93:223-228

56 Bolongese L ,Carabba N Parodi G et alpact of micro vascular dysfunctioion on the left ventricular remodeling and long term clinical outcome after PCI for acute myocardial infarction Circulation 2004;109:2080-2085

57 Shah PK ,Francis GS,Pump failure Shock and Cardiac rupture in acute myocardial infarction in francis GS Alpert JSed Modern Coronary Care Boston Yoronto London : Little Brown co1990;295-325

58 Pfeffer JM, Pfeffer MA, Fletcher PJ, Braunwald E: Progressive ventricular remodeling in rat with myocardial infarction *Am J Physiol* 1991; 260:H1406.

59 Weisman HF, Bush DE, Mannisi JA, et al: Cellular mechanisms of myocardial infarction expansion. *Circulation* 1988; 78:186.

60 Hochman J S,Choo H Limitation of myocardial infarct expansion by reperfusion independent of myocardial salvage Circulation 1987;75:299- 306

61 Forrester JS, Wyatt HL, Da Luz PL, et al: Functional significance of regional ischemic contraction abnormalities. *Circulation* 1976; 54:64.

62 Brown EJ SwinfordRD ,gade P et al Acute effects delayed

reperfusionon myocardial infarct shapeand left ventricular volume a potential mechanism of additional benefits of thrombolytic therapyJ Am Coll Cardiol 991; 17:1641-1650

63 Becker R , Charlesworth A,Wilcox Ret al Cardiac rupture associated with thrombolytic therapy: impact of time to treatment in the Late Assessment of Thrombolytic Efficacy (LATE) study J Am Coll Cardiol1995;25:1063-1068

64 Becker RC Gore JM Lambrew C et alA composite view of cardiac rupture in the United States National Registry of Myocardial Infarction J Am Coll Cardiol1996;27:1321-1326

65 Crenshaw BS ,Granger CB , BirBaum Y Risk factors angiographic pattern and outcomes in patient with VSD complicating acute myocardial infarction Circulation 200;101:27-32

66 Slatter RJ, Brown RJ Antonelli TAetal Cardiogenic shock due to free wall rupture after acute myocardial infarction from SHOCK tial registry J AM Coll Cardiol 2000;36:1117-1122

67 Killip T III , Kimball JT : Treatment of myocardial infarction in coronary care unit; a two year experience with 250 patients Am J Cardiol 20:457,1967

68 Audrey H ,Wu MD,Lori Parsons BS etal , Hospital outcomes in patients presenting with congestive heartfailure complicating acute myocardial infarction :A report from the second National Registry of Myocardial Infarction (NRMI -2) J Am Coll Cardiol 2002 ;40: 1389-94

69 Hochman JS ,Sleeper LA ,Godfrey E et al , for the SHOCK investigators Should we emergently revascularize Occluded Coronaries for cardiogenic shock an international randiomized trial of emergency PTCA /CABG- Trial design Am Heart J 1999;137:313-21

70 Dauerman HL Goldberg RJ White K et al Global Registry of Acute Coronary Events (GRACE) investigators. Revascularisation Stening Outcome of patients with cardiogenic shock complicated by acute myocardial infarction Am J Cardiol 2002;90;838-42

71 Hochman JS ,Sleeper LA , Buller CA et al etiologies management outcome overall findings of SHOCK Trial RegistryCardiogenic shock after acute myocardial infarction *J Am Coll Cardiol* 2000;36:1063-70

72 Goldberg RJ, Gore JM, Alpert JS, et al. Cardiogenic shock after acute myocardial infarction. Incidence and mortality from a community-wide perspective, 1975 to 1988. *N Engl J Med* 1991;325:1117–1122. [PMID: 1891019]

73 Chobanian AV, Bakris GL, Black HR, et al. The seventh report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure: the JNC 7 report. *JAMA* 2003;289(19):2560–2572. [PMID: 12748199]

74 Bayes de Luna A,WagnerG, BirBaum Y etal A new terminology for the leftventricular walland for the locations of Qwave and Q wave equivqlent myocardial infarcts based on the standard of cardiac magneticresonance imaging A statement for the health care professionals from committee appointed by the internationalsocietyfor Holterand non invasive electrocardiography Circulation 2006;114:1755-1760

75 Libby: Braunwald ‘s Heart disease ;A text book of cardiovascular medicine 8th edition 2007 chapter:35,p863-923

76 Crenshaw BS, Ward SR, Granger CB, Stebbins AL, Topol EJ, Califf RM. Atrial fibrillation in the setting of acute myocardial infarction: the GUSTO-I experience. *J Am Coll Cardiol* 1997;30:406–413. [PMID: 9247512]

77 Pedersen OD, Bagger H, Kober L, Torp-Pedersen C. The occurrence and prognostic significance of atrial fibrillation/-flutter following acute myocardial infarction. *Eur Heart J* 1999;20:748–754. [PMID: 10329066]

78 Wong CK ,White HD, Wilcox RG et al New onset atrial fibrillation after acute MI indepedantly predicts mortality GUSTO III experience Am heart J 2000; 140:878- 885

79 Wong CK ,White HD, Wilcox RG et al Management and outcome of patients with atrial fibrillation during acute MI: GUSTO III experience Heart2002; 88: 354 – 6

80 Scine M , Alkhatib MD, MHS et al Sustained ventricular arrhythmias and mortality among patients with acute MI AJCC ; 2003 may :1205-12

81 Volpi A, Cavalli A, Santaro L, et al. Incidence and prognosis of early primary ventricular fibrillation in acute myocardial infarction: results of the Gruppo Italiano per lo Studio della Sopravvivenza nell'Infarto Miocardico (GISSI-2) database. *Am J Cardiol* 1998;82(3):265–271. [PMID: 9708651]

82 Keith H ,Newby ,MD; Trevor Thompson,BS;et al Sustained ventricular arrhythmias in patients receiving thrombolytic therapy incidence and outcomes 1998;98:2567-73

83 GUSTO (Global utilization of Streptokinase & tissue plasminogen

activatorfor occluded coronary arteries) An international RCT comparing four thrombolytic strategies for acute MI N Eng J Med 1993;329:673-682

84 GISSI-2 A factorial randomized trial of alteplase versus streptokinase and heparin versus no heparin among 12490 patients with acute MI.The Lancet 1990;336:65-71

85

Harpaz, D, Behar S, Gottileb S, et al. Complete atrioventricular block complicating acute myocardial infarction in the thrombolytic era. *J Am Coll Cardiol* 1999;34:1721–1728.

86 MelgarejoMarenoA,galcera-Tomas,Garcia AlberolaAetal.Incidence clinical characteristics and prognostic significance of RBBB in acute MI:a study in thrombolytic era Circulation 1997; 96:1139- 44

87 MelgarejoMarenoA,galcera-Tomas,GarciaAlberolaAetal.Incidence clinical characteristics and prognostic significance of LBBB in acute MI:a study in thrombolytic era Rev Esp Cardiol 1999;52:245-52 C

88 NewbyKH, Pisano E, Krokoff MW. Incidence and clinical relevanceof the occurance of bundle branch block in patients with thrombolytic therapy Circulation 1996;94:2424-8

89 Alan S Go MD,Hal V et al Bundle branch block and in hospital

mortality in acute myocardial infarction .

Annals of Int Medicine 1998;vol129: (9)690-97

PROFORMA

PROGNOSTIC SIGNIFICANCE OF ARRHYTHMIAS IN ST ELEVATION MYOCARDIAL INFARCTION

Name Age Sex

Lifestyle

Smoker Alcoholic

DM SHT

CRF

SYMPTOMS :

Chest pain

Dyspnoea Others

Palpitation

Syncope

EXAMINATION:

BMI Pallor JVP

Pulse BP

CVS : RS

Electrocardiogram :

Rate

Regional wall

Reperfusion Status

ARRHYTHMIA

OUTCOME

ECHOCARDIOGRAM
RWMA
EF (%)
Others

HEART FAILURE
(Killip class)
SHOCK
DIED

FOLLOW UP: